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=> s p53 and antisense
L1         4478 P53 AND ANTISENSE

=> s l1 and splice acceptor site
L2           78 L1 AND SPLICE ACCEPTOR SITE

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           78 DUP REM L2 (0 DUPLICATES REMOVED)

=> s l3 and morpholino
L4            2 L3 AND MORPHOLINO

=> d l4 ibib abs tot

L4   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2001:816897  CAPLUS
DOCUMENT NUMBER:          135:353717
TITLE:                    Splice-region   ***antisense***   oligonucleotide
                          composition and targeting the mRNA splicing
INVENTOR(S):              Iversen, Patrick L.; Hudziak, Robert
PATENT ASSIGNEE(S):       Avi Biopharma, Inc., USA
SOURCE:                   PCT Int. Appl., 53 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2001083740      A2   20011108       WO 2001-US14410  20010504
         W:  AU, CA, JP, KR
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, TR
PRIORITY APPLN. INFO.:                    US 2000-202376P  P  20000504
AB      ***Antisense***   compns. targeted against an mRNA sequence for a
     selected protein, at a region having its 5' end from 1 to about 25 base
     pairs downstream of a normal splice acceptor junction in the preprocessed
     mRNA, are disclosed.  The   ***antisense***   compd. is Rnase-inactive,
     and is preferably a phosphorodiamidate-linked   ***morpholino***
     oligonucleotide.  Such targeting is effective to inhibit natural mRNA
     splice processing, produce splice variant mRNAs, and inhibit normal
     expression of the protein.

L4   ANSWER 2 OF 2  USPATFULL
ACCESSION NUMBER:         2000:57750  USPATFULL
TITLE:                    Chimeric oligonucleoside compounds
INVENTOR(S):              Arnold, Jr., Lyle J., Poway, CA, United States
```

Giachetti, Cristina, Solano Beach, CA, United States
Lebedev, Alexandre V., San Diego, CA, United States

PATENT ASSIGNEE(S): Genta Incorporated, Lexington, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6060456		20000509
APPLICATION INFO.:	US 1997-960111		19971027 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-481637, filed on 7 Jun 1995, now abandoned which is a continuation of Ser. No. US 1994-238177, filed on 4 May 1994, now abandoned which is a continuation of Ser. No. US 1994-233778, filed on 26 Apr 1994, now abandoned which is a continuation of Ser. No. US 1993-154013, filed on 16 Nov 1993, now abandoned which is a continuation of Ser. No. US 1993-154014, filed on 16 Nov 1993, now abandoned

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Knobbe, Martens, Olson & Bear LLP.
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 12 Drawing Page(s)
LINE COUNT: 5081
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Chimeric oligonucleoside compounds, and methods of preparing and formulating the same, are disclosed. The compounds and compositions are useful in activating RNaseH-mediated cleavage of target ribonucleic acid sequences, and in treating disease conditions relating to such sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l4 2 kwic

L4 ANSWER 2 OF 2 USPATFULL

SUMM The present invention relates to ***antisense*** oligonucleoside compounds containing modified internucleoside linkages, and optionally other structural modifications. The compounds are capable of hybridizing to target nucleic. . .

SUMM Considerable attention has been directed in recent years to the design of ***antisense*** nucleic acid oligomers for use in studying, treating and diagnosing conditions attributable to endogenous or foreign nucleic acid sequences in living organisms. For example, it is now well known that a nucleic acid oligomer having suitable ***antisense*** complementarity to a target mRNA can hybridize to the target mRNA and, in some cases, disrupt translation of the mRNA. The ***antisense*** approach presents great promise for the eventual therapeutic treatment of disease conditions caused by foreign (e.g., viral) genetic material, or. . .

SUMM However, despite the great promise of the ***antisense*** approach, a number of challenges still remain. First, ***antisense*** compounds are generally subject to degradation in the cellular milieu due to endogenous endo- and exonucleases. While a number of modified ***antisense*** structures have been described having improved resistance to nuclease degradation, further improvements are desirable in order to increase the potency and half-life of the compounds. Second, it is generally required that an ***antisense*** compound have a high specificity toward the intended target nucleic acid so as to avoid disruption of activity of unintended native sequences. Although a number of researchers have described approaches designed to increase the binding affinity of an ***antisense*** compound to a target sequence, very few results have been reported with respect to structural refinements which avoid disruption of. . .

SUMM . . . approach toward disrupting the expression of undesired target mRNAs involves forming a duplex hybrid between the target mRNA and an ***antisense*** strand, followed by cleavage of the target mRNA by an endogenous RNaseH. See Dash, P., et al, Proc. Natl. Acad.. . . Nucleases (Linn & Roberts, eds.), Cold Spring Harbor Laboratory (1982), at 212. As a result, one putative requirement of the ***antisense*** RNaseH cleavage approach is that at least some of the nucleosides of the ***antisense*** nucleic acid strand must have characteristics in common with deoxyribonucleotides (as opposed to ribonucleotides), particularly, the absence of a polar group on the 2'-position of the

additional requirement that at least some of the sugar groups in the ***antisense*** compound must be in a 2'-endo (.beta.) conformation as found in deoxyribonucleosides, as opposed to the 3'-endo (.alpha.) conformation found. . .

SUMM It has further been reported that various 2'-position substituents (e.g., 2'-O-alkyl and 2'-fluoro) will render the substituted portion of an ***antisense*** strand non-activating to RNaseH, even though binding affinity toward the target nucleic acid is increased. Inoue, H., et al., FEBS. . . achieve efficient activation of mammalian (HeLa) RNAseH, and that this 2'-deoxy segment (if accompanied by 2'-substituted residues in the same ***antisense*** compound) must be centered in the oligomer sequence in order to achieve efficient RNaseH activation in vitro or expression inhibition. . .

SUMM Another reported requirement of the ***antisense*** RNaseH cleavage approach is that, in order to achieve RNaseH activation, at least one portion of the internucleoside "backbone" of the ***antisense*** compound must include charged (anionic) phosphorus-containing linkage groups. Cook, P. D., PCT Publication No. WO 93/13121 (1993), at 18. In studies of chimeric ***antisense*** compounds including both methylphosphonate (uncharged) and phosphodiester or phosphorothioate (charged) linkages, Agrawal, et al. reported that the minimum number of. . .

SUMM . . . exonucleases. A variety of alternative linkage groups, some of which are nuclease-resistant, have been developed or proposed for use with ***antisense*** compounds. Among these are charged linkage groups such as phosphorothioate, phosphorodithioate, phosphoroselenate and phosphorodiselenate linkers. In general, deoxyribonucleoside ***antisense*** oligomers containing these non-natural linkage groups tend to have lower binding affinity toward complementary RNA target strands than the corresponding phosphodiester-linked ***antisense*** oligomers, although higher affinity may be achieved where the ***antisense*** strand comprises ribonucleosides or 2'-substituted ribonucleosides (rather than deoxyribonucleosides). See Metelev, V. & Agrawal, S., PCT Publication No. WO 94/02498. . . Padmapriya, A. & Agrawal S., PCT Publication No. WO 94/02499 (1994). Non-phosphorus-based linkage groups have also been reported, including peptide, ***morpholino*** , ethylene glycol, amide, and other linkers. See Reynolds, M. A., et al., PCT Publication No. WO 92/02532 (1992); Cook, P.. . . lower binding affinity (compared to phosphodiester linkages) toward complementary RNA target strands, at least in the case of linked 2'-unsubstituted ***antisense*** nucleotides, and particularly in the presence of salt ions.

SUMM Various workers have attempted to identify combinations of linkage groups and/or structural modifications for ***antisense*** oligomers that might lead to improved RNaseH activation, binding affinity, nuclease resistance and/or target specificity. Thus, Cohen, et al. have reported improved half-life for ***antisense*** and non-***antisense*** oligodeoxyribonucleotides containing at least one phosphorothioate linkage located, for example, at either terminus of the compound, or throughout the compound.. . . RNaseH cleavage activation reportedly required retention of at least four, and preferably at least seven, contiguous phosphodiester linkages in the ***antisense*** oligomer. The preferred compounds contained at least 10, and preferably at least 15, nucleotides, the majority of which were phosphodiester-linked.. . .

SUMM Giles & Tidd have reported that the target specificity of an ***antisense*** oligomer can be improved by the use of a chimeric structure comprising terminal methylphosphonodiester sections separated by a central RNaseH-activating. . .

SUMM . . . was reportedly localized to a site (or sites) on the target corresponding to the non-substituted (i.e., deoxyribonucleotide) portion of the ***antisense*** compound. Single-site cleavage was reportedly optimized by use of a tetradeoxyribonucleotide segment located centrally in the compound between two 2'-substituted. . .

SUMM The present invention relates to improved RNaseH-activating ***antisense*** oligonucleoside compounds containing selectively modified internucleoside linkages, and optionally other structural modifications. The compounds exhibit improved target specificity and potency compared to other RNaseH-activating ***antisense*** compounds. They are useful both in vivo and in vitro in reducing or eliminating the translation of target mRNA sequences,. . .

SUMM In another aspect, the present invention provides chimeric structures for ***antisense*** oligonucleoside compounds that maximize activity while maintaining the ability to effect selective RNaseH-mediated cleavage of the intended target strand. These. . .

SUMM In another aspect, the present invention includes imp roved

diagnosing diseases or other conditions in living organisms attributable to the expression of endogenous. . .

DETD Consider first the challenge of achieving high target specificity with an ***antisense*** cleavage compound. Mammalian cells typically contain an RNA population comprising about 3.times.10.sup.7 ribonucleotides. By assuming a statistically random distribution of. . .

DETD . . . mismatches increases. If the K.sub.A for a given mismatch duplex is sufficiently high as to allow appreciable hybridization of an ***antisense*** oligomer to a mismatched target, then unintended and undesirable cleavage of the mismatched target can result.

DETD Take, for example, the case of a one-base mismatch between a 12-to-18 nucleoside ***antisense*** oligomer and an unintended mismatch RNA sequence. The present inventors have ascertained that the K.sub.A for the correct "match" hybridization. . .

DETD . . . and polyadenylation sites), inhibition of protein production can be achieved prior to the translation process by suitable hybridization of an ***antisense*** oligonucleoside, and ribosomal displacement of the hybridized oligomer generally does not occur. As a result, oligonucleosides having higher binding affinities. . .

DETD . . . reviewed by Jaeger et al., Annual Reviews in Biochemistry 62, 255-287 (1993). Another approach is to utilize two or more ***antisense*** compounds in tandem, at least one of which is a chimeric oligonucleoside of the invention, which ***antisense*** compounds have nucleoside base sequences selected to hybridize to adjacent regions in a secondary-structured mRNA target region. It is known that adjacently-hybridizing ***antisense*** compounds may be used to disrupt secondary structure of RNA molecules and thus to enhance the effective K.sub.A 's of. . .

DETD As discussed above in the background section of this disclosure, a number of workers in the ***antisense*** field have reported various and disparate efforts to increase binding affinity of ***antisense*** oligonucleosides, to optimize RNaseH activation, to improve nuclease resistance, and to improve target specificity. It will be seen in light. . .

DETD As is also explained above, one putative requirement of mammalian RNaseH activation is that the ***antisense*** compound must have a sequence of at least four or five consecutive charged (anionic) internucleoside linkage structures (or at least. . .

DETD The present invention likewise includes chimeric ***antisense*** oligomers having enhanced potency as ***antisense*** inhibitors of gene expression comprising one or more segments with methylphosphonate internucleosidyl linkages enhanced for the R.sub.p configuration which are. . .

DETD . . . originally derived from the coupled dimeric units). The remaining internucleosidyl linkages comprise non-phosphonate internucleosidyl linkages, such as phosphodiester, phosphorothioate, phosphorodithioate, ***morpholino*** , phosphoramidite, phosphorofluoridate, boranophosphate, formacetal, silyl or other non-phosphonate internucleosidyl linkages.

DETD . . . that the sequence of nucleoside bases in the present chimeric RNaseH-activating compounds, and also the sequence of bases in RNaseH-activating ***antisense*** compounds generally, can be selected in a manner described herein to provide RNaseH-activated cleavage that is highly specific to disease. . . caused by singular (particularly single-base) mutations, allele differences or other anomalies in genetic sequence. In particular, the inventors have discovered ***antisense*** oligonucleoside constructions that are capable of discriminating a single base difference in a target RNA sequence occurring at a base. . .

DETD In this regard, ***antisense*** oligonucleosides have reportedly been used to target a wide variety of mRNAs associated with cancer and other diseases. See, for. . . Science, 261, 1004-1012 (1993); Uhlmann, E. and Peyman, A., Chemical Reviews, 90, 544-584 (1990). One challenge in the application of ***antisense*** oligonucleosides as therapeutic agents is the need to discriminate a single base difference between target and non-target sequences. A number. . . for example, have been identified which differ from their normal counterparts by only a single base change, including the RAS, ***p53*** , src and erbB-2 oncogenes. Single base changes are also associated with some inherited genetic diseases such as Lesch-Nyhan syndrome (Fujimura,. . .

DETD Ideally, and within the constraints described in the specification above, ***antisense*** oligonucleosides should have high affinities with their target sequences, so that small amounts of oligonucleoside can be used for treatment.. . .

DETD . . . oligomers can be achieved by carefully defining the position of

antisense oligomer. Particularly, the oligomer is selected, first, to have a base sequence that is complementary to a target region of. . .

DETD . . . hypotonic dounce lysis in 5.times. the packed cell volume. It was buffered to pH 6.0 by adding 0.4 mL of 2-(N- ***morpholino***) ethanesulfonate (MES, 0.5 M solution, pH 6.0) to 3.6 mL of cell lysate on ice and mixing with mild agitation.. . .

DETD Inhibition of Protein Synthesis in a Cell Culture With Chimeric ***Antisense*** Oligomers Targeted to a Non-Eukaryotic Reporter Gene, Chloramphenicol Transferase

DETD The following example shows the ability of chimeric ***antisense*** oligomers to selectively inhibit protein synthesis in a eukaryotic cell culture system. COS-7 cells were transiently transfected with plasmids encoding either a target reporter gene or a control non-target reporter gene. These cells were then treated with various chimeric ***antisense*** or control oligomers and then assayed for the expression of the reporter genes.

DETD . . . tct gca 3'

XV-6 SEQ ID NO:9,
24mer, all phosphorothioate:
5' cac tca atc aat gac tag tct gca 3'
Splice ***acceptor*** ***site*** oligomers:

3265-1 SEQ ID NO:19,
24mer, (MP(R.sub.p)/DE)(PS/DE)(MP(R.sub.p)/DE):
5' ccc tga ga(g aga g)ag aga ggt tcg 3'

3266-1 SEQ ID. . .

DETD Anti-Splice Site Oligomers Versus pG1035 and pG1036 (splicing inhibition by ***antisense*** oligomers):

DETD . . . splice site acceptor oligomer 3387-1 and lengthening the PS center from five to seven continuous phosphorothioate backbone linkages increases the ***antisense*** activity against the ***splice*** ***acceptor*** ***site*** target significantly but does not increase non-specific activity against the control target.

DETD . . . all-PS oligomers and control chimeras. Both target-specific and oligomer-specific controls were included, demonstrating that the results are based on sequence-specific ***antisense*** effects.

DETD . . . of an oligonucleoside and/or in the target mRNA. The chimeric compounds listed below (see also Example 41) were assayed for ***antisense*** activity against both the pG1040 (UCAT) target and the pG1042 (UCAT) 4-base mismatch control. The oligomer sequences were as follows.

DETD pG1040 (UCAT) target mRNA and ***antisense*** oligomers:

+1 +4 +27
.vertline. .vertline. .vertline.
Met Glu Lys Lys Ile Ser Gly Tyr Thr ... ...

mRNA .... . .

DETD ***Antisense*** activity was assayed against both pG1041 (UCAT) and pG1042 (UCAT) using procedures as generally described in Example 41, except that. . . It was demonstrated that mismatches in the phosphorothioate core and the position of the core in chimeric oligomers greatly affected ***antisense*** activity. The following table sets forth the percentage of gene expression (.+-.error) measured for each of the tested oligomers.

DETD . . . oligomer. The position of the phosphorothioate core and/or the base composition of the phosphorothioate core has a large effect on ***antisense*** activity, as seen by comparing 3637-1, 3638-1, 3262-5 and 3636-1. A more central position within the chimera is most active,. . .

DETD . . . (denoted by an "x" above the sequences shown above) within the RNaseH phosphorothioate core sequence of the chimeric oligomers eliminates ***antisense*** activity in this eukaryotic cell culture assay, as seen by comparing 3639-1 and 3640-1 with 3262-5. In a separate experiment. . .

DETD D. Demonstration of Activity of ***Antisense*** Chimeric Oligomers Targeted to HPV-11 E7 in Cell Free Translation Extracts

DETD E. Demonstration of Activity of ***Antisense*** Oligomers in a Cell-Free RNAseH Cleavage Assay

DETD F. Demonstration of Activity of ***Antisense*** Oligomers in Transiently Transfected COS-7 Cells

DETD Representative experiments were performed as follows. E7 expression plasmid pcDNA11E7 (5 .mu.g/ml) and different amounts of ***antisense*** oligonucleotide were transfected into COS-7 cells in the presence of Transfectam.TM. (Promega). Cells were incubated with transfection mixture for 4. . .

DETD H. Demonstration of Activity of ***Antisense*** Oligomers Targeted to E2 in Cell-Free Translation Extracts

to E6 in Cell-Free Translation Extracts

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=> d history

     (FILE 'HOME' ENTERED AT 19:01:02 ON 21 JAN 2003)

     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     19:01:24 ON 21 JAN 2003
L1          4478 S P53 AND ANTISENSE
L2            78 S L1 AND SPLICE ACCEPTOR SITE
L3            78 DUP REM L2 (0 DUPLICATES REMOVED)
L4             2 S L3 AND MORPHOLINO

=> s l1 and morpholino
L5          171 L1 AND MORPHOLINO

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6           157 DUP REM L5 (14 DUPLICATES REMOVED)

=> s l6 and py<2001
   3 FILES SEARCHED...
L7           37 L6 AND PY<2001

=> d l7 ibib abs tot

L7   ANSWER 1 OF 37     MEDLINE
ACCESSION NUMBER:    2000153646     MEDLINE
DOCUMENT NUMBER:     20153646   PubMed ID: 10688605
TITLE:               c-Myc   ***antisense***   limits rat liver regeneration and
                     indicates role for c-Myc in regulating cytochrome P-450 3A
                     activity.
AUTHOR:              Arora V; Knapp D C; Smith B L; Statdfield M L; Stein D A;
                     Reddy M T; Weller D D; Iversen P L
CORPORATE SOURCE:    AVI BioPharma, Corvallis, Oregon, USA.
CONTRACT NUMBER:     GM54871 (NIGMS)
SOURCE:              JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS,
                       ***(2000 Mar)***   292 (3) 921-8.
                     Journal code: 0376362. ISSN: 0022-3565.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200003
ENTRY DATE:          Entered STN: 20000330
                     Last Updated on STN: 20000330
                     Entered Medline: 20000317
AB   Expression of c-myc protein is associated with cell proliferation. The
     present study uses   ***antisense***   oligomers to inhibit c-myc
     expression in the regenerating rat liver after 70% partial hepatectomy
     (PH).   ***Antisense***   phosphorodiamidate   ***morpholino***
     oligomers (novel DNA analogs) were administered i.p. immediately after
     surgery to block expression of c-myc within the first 24 h after PH. A
     20-mer PMO complimentary to the c-myc mRNA at the translation start site
     was an effective sequence (AVI-4126, 5'-ACGTTGAGGGGCATCGTCGC-3'). A single
     i.p. dose of 0.5 mg/kg AVI-4126 caused reduction of the regenerating liver
     c-myc protein in a sequence-specific and dose-dependent manner. Inhibition
     of c-myc expression resulted in reduction of proliferating cell nuclear
     antigen and arrested cells in the G(0)/G(1) phase of the cell cycle. The
     ratio of G(2):G(0) cell populations in the regenerating liver 24 h after
     PH dropped from 29.1 in saline vehicle-treated rats to 18.0 in rats
     treated with 2.5 mg/kg AVI-4126. The expression of cell cycle checkpoint
     protein   ***p53***   was inhibited with increasing doses of AVI-4126, but
     expression of p21(waf-1) was unaffected. The activity of cytochrome P-450
     3A2 (CYP3A2) was evaluated by immunoblot analysis and erythromycin
     N-demethylation. AVI-4126 did not alter CYP3A activity in
     nonhepatectamized animals but showed a dose-dependent decrease in PH rats.
     We conclude that AVI-4126,   ***antisense***   oligomer to c-myc, can
     reduce cell proliferation in the regenerating rat liver. Furthermore,
     inhibition of c-myc may indirectly influence the expression of CYP3A.

L7   ANSWER 2 OF 37  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:        2000:291227  CAPLUS
DOCUMENT NUMBER:         132:318574
TITLE:                     ***p53***  -targeted   ***antisense***
```

cancer and hypoxia-induced disorders
INVENTOR(S): Iversen, Patrick L.
PATENT ASSIGNEE(S): Avi Biopharma, Inc., USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000024885	A2	20000504	WO 1999-US24758	19991022 <--
WO 2000024885	A3	20000720		
W: AU, CA, JP, KR				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1124950	A2	20010822	EP 1999-971033	19991022
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
US 6365577	B1	20020402	US 1999-426804	19991022

PRIORITY APPLN. INFO.: US 1998-105695P P 19981026
WO 1999-US24758 W 19991022

AB ***Antisense*** oligonucleotides useful for treating a disease state characterized by ***p53*** induction, such as proliferative cell disorders, e.g. cancer, or a hypoxic state induced by an ischemic attack, such as stroke, are described. The ***antisense*** agents are preferably of the class known as "steric blocker" type oligonucleotides, including ***morpholino*** oligonucleotides, peptide nucleic acids, 2' O-allyl or 2'-O-alkyl modified oligonucleotides, or N3' <<rwa P5' phosphoramidate oligonucleotides. Thus, in partially hepatectomized rats, ***morpholino*** - or C5-propyne cytosine-contg. ***p53*** ***antisense*** oligonucleotides enhanced wt. gain in the regenerating livers.

L7 ANSWER 3 OF 37 USPATFULL
ACCESSION NUMBER: 2000:157559 USPATFULL
TITLE: Modified oligonucleotides, their preparation and their use
INVENTOR(S): Seela, Frank, Osnabruck, Germany, Federal Republic of
Thomas, Horst, Hasbergen, Germany, Federal Republic of
PATENT ASSIGNEE(S): Aventis Pharma Deutschland GmbH, Frankfurt am Main, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6150510		20001121	<--
APPLICATION INFO.:	US 1998-144112		19980831 (9)	

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-554164, filed on 6 Nov 1995, now patented, Pat. No. US 5844106
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Foley & Lardner
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
LINE COUNT: 3592
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Modified oligonucleotides which possess at least one substituted 7-deazapurine base form more stable hybridization complexes with nucleic acids than unsubstituted analogs. They are useful as inhibitors of gene expression, as probes for detecting nucleic acids, as aids in molecular biology and as pharmaceuticals or diagnostic agents. Processes for preparing them are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 4 OF 37 USPATFULL
ACCESSION NUMBER: 2000:138328 USPATFULL
TITLE: Inhibition of extracellular matrix synthesis by ***antisense*** compounds directed to nuclear proto-oncogenes
INVENTOR(S): Zalewski, Andrew, Elkins Park, PA, United States
Shi, Yi, Cheltenham, PA, United States
PATENT ASSIGNEE(S): Thomas Jefferson Univerisity, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6133242		20001017	<--
APPLICATION INFO.:	US 1995-461366		19950605 (8)	

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 1994-US11853, filed on 17 Oct 1994 which is a continuation-in-part of Ser. No. US 1993-138637, filed on 15 Oct 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Brusca, John S.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Seidal, Gonda, Lavorgna & Monaco, PC
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 32 Drawing Figure(s); 23 Drawing Page(s)
LINE COUNT: 2109
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method and compounds are provided for inhibiting the synthesis of extracellular matrix proteins. Compounds of the invention comprise ***antisense*** oligonucleotides specific for nuclear proto-oncogenes. Preferably, ***antisense*** compounds of the invention are selected from the group consisting of c-myc and c-myb and are locally administered. The invention finds use in the treatment of a variety of disorders, including sclerotic disorders and restenosis, associated with the inappropriate synthesis of extracellular matrix proteins, particularly collagen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 5 OF 37 USPATFULL
ACCESSION NUMBER: 2000:124778 USPATFULL
TITLE: Antitumor ***antisense*** sequences directed against R1 and R2 components of ribonucleotide reductase
INVENTOR(S): Wright, Jim A., Toronto, Canada
Young, Aiping H., Toronto, Canada
PATENT ASSIGNEE(S): Genesense Technologies, Inc., Toronto, Canada (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6121000		20000919	<--
APPLICATION INFO.:	US 1999-249730		19990211 (9)	

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-23040P	19960802 (60)
	US 1997-39959P	19970307 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Guzo, David
ASSISTANT EXAMINER: Wang, Andrew
LEGAL REPRESENTATIVE: Burns, Doane, Swecker & Mathis, LLP
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 4
NUMBER OF DRAWINGS: 23 Drawing Figure(s); 23 Drawing Page(s)
LINE COUNT: 4251
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compounds and methods for modulating cell proliferation, preferably inhibiting the proliferation of tumor cells are described. Compounds that may be used to modulate cell proliferation include ***antisense*** oligonucleotides complementary to regions of the mammalian ribonucleotide reductase genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 6 OF 37 USPATFULL
ACCESSION NUMBER: 2000:109600 USPATFULL
TITLE: Oligoribonucleotides and ribonucleases for cleaving RNA
INVENTOR(S): Crooke, Stanley T., Carlsbad, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6107094		20000822	<--

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-659440, filed on 6 Jun 1996, now patented, Pat. No. US 5898031
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz Mackiewicz & Norris LLP
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 3806
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Oligomeric compounds including oligoribonucleotides and oligoribonucleosides are provided that have subsequences of 2'-pentoribofuranosyl nucleosides that activate dsRNase. The oligoribonucleotides and oligoribonucleosides can include substituent groups for increasing binding affinity to complementary nucleic acid strand as well as substituent groups for increasing nuclease resistance. The oligomeric compounds are useful for diagnostics and other research purposes, for modulating the expression of a protein in organisms, and for the diagnosis, detection and treatment of other conditions susceptible to oligonucleotide therapeutics. Also included in the invention are mammalian ribonucleases, i.e., enzymes that degrade RNA, and substrates for such ribonucleases. Such a ribonuclease is referred to herein as a dsRNase, wherein "ds" indicates the RNase's specificity for certain double-stranded RNA substrates. The artificial substrates for the dsRNases described herein are useful in preparing affinity matrices for purifying mammalian ribonuclease as well as non-degradative RNA-binding proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 7 OF 37 USPATFULL
ACCESSION NUMBER: 2000:98562 USPATFULL
TITLE: Circular DNA vectors for synthesis of RNA and DNA
INVENTOR(S): Kool, Eric T., Rochester, NY, United States
PATENT ASSIGNEE(S): University of Rochester, Rochester, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6096880		20000801	<--
APPLICATION INFO.:	US 1997-805631		19970226 (8)	

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-393439, filed on 23 Feb 1995, now patented, Pat. No. US 5714320 which is a continuation-in-part of Ser. No. US 1993-47860, filed on 15 Apr 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliot, George C.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Mueting, Raasch & Gebhardt, P.A.
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 3103
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides methods for synthesis, and therapeutic use of DNA and RNA oligonucleotides and analogs. RNA oligonucleotides arc synthesized using a small, circular DNA template which lacks an RNA polymerase promoter sequence. The RNA synthesis is performed by combining a circular single-stranded oligonucleotide template with an effective RNA polymerase and at least two types of ribonucleotide triphosphate to form an RNA oligonucleotide multimer comprising multiple copies of the desired RNA oligonucleotide sequence. Preferably, the RNA oligonucleotide multimer is cleaved to produce RNA oligonucleotides having well-defined ends. Preferred RNA oligonucleotide multimers contain ribozymes capable of both cis (autolytic) and trans cleavage.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 8 OF 37 USPATFULL
ACCESSION NUMBER: 2000:95115 USPATFULL
TITLE: Cationic lipids
INVENTOR(S): Lin, Kuei-Ying, Fremont, CA, United States
Mattuecci, Mark D., Burlingame, CA, United States

(U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6093816		20000725	<--
APPLICATION INFO.:	US 1996-672206		19960627	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Higel, Floyd D.
LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz Mackiewicz & Norris LLP
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
LINE COUNT: 2544
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to new cationic lipids and intermediates in their synthesis that are useful for transfecting nucleic acids or peptides into prokaryotic or eukaryotic cells. The lipids comprise one or two substituted histidine residues, or similar compounds, linked to a lipophilic moiety. The lipids form a complex when mixed with polyanions such as nucleic acids or peptides. The complexes permit efficient transfer of polyanions into cells, usually without significant toxicity to the cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 9 OF 37 USPATFULL
ACCESSION NUMBER: 2000:91721 USPATFULL
TITLE: Diagnostic method detecting loss of wild-type ***p53***
INVENTOR(S): Vogelstein, Bert, Baltimore, MD, United States
Baker, Suzanne J., Baltimore, MD, United States
Fearon, Eric R., Baltimore, MD, United States
Nigro, Janice M., Baltimore, MD, United States
PATENT ASSIGNEE(S): Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6090566		20000718	<--
APPLICATION INFO.:	US 1995-459676		19950602	(8)

RELATED APPLN. INFO.: Division of Ser. No. US 1993-47041, filed on 22 Mar 1993, now patented, Pat. No. US 5527676 And a continuation of Ser. No. US 1992-928661, filed on 17 Aug 1992, now abandoned And a continuation of Ser. No. US 1989-446584, filed on 6 Dec 1989, now abandoned And a continuation-in-part of Ser. No. US 1989-330566, filed on 29 Mar 1989, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Eyler, Yvonne
LEGAL REPRESENTATIVE: Banner & Witcoff, Ltd.
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1011
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and kits are provided for assessing mutations and/or loss of the ***p53*** gene in human tumors. Both deletion mutations and point mutations in ***p53*** are observed in the same human tumor cells and these mutations are clonal within the cells of the tumor. Loss of wild-type ***p53*** genes is responsible for neoplastic progression.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 10 OF 37 USPATFULL
ACCESSION NUMBER: 2000:77184 USPATFULL
TITLE: Highly sensitive multimeric nucleic acid probes
INVENTOR(S): Kool, Eric T., Rochester, NY, United States
PATENT ASSIGNEE(S): University of Rochester, Rochester, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6077668		20000620	<--
APPLICATION INFO.:	US 1997-910632		19970813	(8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-805631, filed

Feb 1995, now patented, Pat. No. US 5714320, issued on 3 Feb 1998 which is a continuation-in-part of Ser. No. US 1993-47860, filed on 15 Apr 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Brusca, John S.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Mueting, Raasch & Gebhardt, P.A.
NUMBER OF CLAIMS: 66
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 3477
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides detectably labeled RNA and DNA oligonucleotide multimers useful as diagnostic probes in medical, biological and chemical applications. A method for synthesizing DNA and RNA oligonucleotides, oligonucleotide multimers, and analogs, preferably those that are detectably labeled, is also provided. Oligonucleotide synthesis is performed by combining a circular single-stranded oligonucleotide template with an effective polymerase and at least two types of nucleotide triphosphate, without the addition of auxiliary proteins, to yield an oligonucleotide multimer comprising multiple copies of a repeated oligonucleotide sequence.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 11 OF 37 USPATFULL
ACCESSION NUMBER: 2000:64945 USPATFULL
TITLE: Modified oligonucleotides, their preparation and their use
INVENTOR(S): Seela, Frank, Osnabruck, Germany, Federal Republic of
Lampe, Sigrid, Berge/Hekese, Germany, Federal Republic of
PATENT ASSIGNEE(S): Hoechst Aktiengesellschaft, Frankfurt am Main, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6066720		20000523	<--
APPLICATION INFO.:	US 1998-94405		19980610 (9)	

RELATED APPLN. INFO.: Division of Ser. No. US 1997-940196, filed on 29 Sep 1997, now patented, Pat. No. US 5789562 which is a continuation of Ser. No. US 1995-431777, filed on 1 May 1995, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1994-4415370	19940502

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
LINE COUNT: 2037
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel modified oligonucleotides which contain at least one 8-azapurine base and form more stable hybridization complexes with nucleic acids; To a process for their preparation, and to their use as inhibitors of gene expression, as probes for detecting nucleic acids, as aids in molecular biology, and as a pharmaceutical or diagnostic agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 12 OF 37 USPATFULL
ACCESSION NUMBER: 2000:57750 USPATFULL
TITLE: Chimeric oligonucleoside compounds
INVENTOR(S): Arnold, Jr., Lyle J., Poway, CA, United States
Reynolds, Mark A., San Diego, CA, United States
Giachetti, Cristina, Solano Beach, CA, United States
Lebedev, Alexandre V., San Diego, CA, United States
PATENT ASSIGNEE(S): Genta Incorporated, Lexington, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION: US 6060456 20000509 <--
APPLICATION INFO.: US 1997-960111 19971027 (8)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-481637, filed on 7 Jun 1995, now abandoned which is a continuation of Ser. No. US 1994-238177, filed on 4 May 1994, now abandoned which is a continuation of Ser. No. US 1994-233778, filed on 26 Apr 1994, now abandoned which is a continuation of Ser. No. US 1993-154013, filed on 16 Nov 1993, now abandoned which is a continuation of Ser. No. US 1993-154014, filed on 16 Nov 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Knobbe, Martens, Olson & Bear LLP.
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 12 Drawing Page(s)
LINE COUNT: 5081
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Chimeric oligonucleoside compounds, and methods of preparing and formulating the same, are disclosed. The compounds and compositions are useful in activating RNaseH-mediated cleavage of target ribonucleic acid sequences, and in treating disease conditions relating to such sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 13 OF 37 USPATFULL
ACCESSION NUMBER: 2000:41165 USPATFULL
TITLE: ***Antisense*** modulation of MDMX expression
INVENTOR(S): Monia, Brett P., La Costa, CA, United States
Cowsert, Lex M., Carlsbad, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6046320		20000404	<--
APPLICATION INFO.:	US 1999-289267		19990409 (9)	

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: Epps, Janet
LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
LINE COUNT: 3298
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB ***Antisense*** compounds, compositions and methods are provided for modulating the expression of MDMX. The compositions comprise ***antisense*** compounds, particularly ***antisense*** oligonucleotides, targeted to nucleic acids encoding MDMX. Methods of using these compounds for modulation of MDMX expression and for treatment of diseases associated with expression of MDMX are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 14 OF 37 USPATFULL
ACCESSION NUMBER: 2000:1692 USPATFULL
TITLE: Sequence-directed DNA binding molecules compositions and methods
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
Turin, Lisa M., Redwood City, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6010849		20000104	<--
APPLICATION INFO.:	US 1995-482080		19950607 (8)	

RELATED APPLN. INFO.: Division of Ser. No. US 1993-171389, filed on 20 Dec 1993, now patented, Pat. No. US 5578444 which is a continuation-in-part of Ser. No. US 1993-123936, filed

is a continuation-in-part of Ser. No. US 1992-996783, filed on 23 Dec 1992, now patented, Pat. No. US 5693463 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degen, Nancy
ASSISTANT EXAMINER: Schwartzman, Robert
LEGAL REPRESENTATIVE: Fabin, Gary R.Dehlinger & Associates
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Figure(s); 47 Drawing Page(s)
LINE COUNT: 10022
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines a DNA:protein-binding assay useful for screening libraries of synthetic or biological compounds for their ability to bind DNA test sequences. The assay is versatile in that any number of test sequences can be tested by placing the test sequence adjacent to a defined protein binding screening sequence. Binding of molecules to these test sequence changes the binding characteristics of the protein molecule to its cognate binding sequence. When such a molecule binds the test sequence the equilibrium of the DNA:protein complexes is disturbed, generating changes in the concentration of free DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ ID NO:600) are set forth. The assay of the present invention is also useful to characterize the preferred binding sequences of any selected DNA-binding molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 15 OF 37 USPATFULL
ACCESSION NUMBER: 1999:159999 USPATFULL
TITLE: Antitumor ***antisense*** sequences directed against ribonucleotide reductase
INVENTOR(S): Wright, Jim A., 15 Bryn Mawr Road, Winnipeg, Manitoba, Canada R3T 3K8
Young, Aiping H., 717 Pacific Avenue, Winnipeg, Manitoba, Canada R3E 1G1

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5998383		19991207	<--
APPLICATION INFO.:	US 1997-904901		19970801	(8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-23040P	19960802 (60)
	US 1997-39959P	19970307 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: LeGuyader, John L.
ASSISTANT EXAMINER: Shibuya, Mark L.
LEGAL REPRESENTATIVE: Burns, Doane, Swecker & Mathis, L.L.P.
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 4530
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A synthetic ***antisense*** oligonucleotide comprising at least seven nucleotides or nucleotide analogues having a sequence complementary to the mRNA sequence of ribonucleotide reductase dimeric protein component R2 including SEQ ID Nos:1-102 is disclosed. A synthetic ***antisense*** oligonucleotide comprising at least seven nucleotides or nucleotide analogues having a sequence complementary to the mRNA sequence of ribonucleotide reductase dimeric protein component R1 including SEQ ID Nos:103-161 is also disclosed. The invention also discloses pharmaceutical compositions including the synthetic ***antisense*** oligonucleotides of the present invention and methods of using the ***antisense*** oligonucleotides to modulation proliferative cells including neoplastic cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 16 OF 37 USPATFULL
ACCESSION NUMBER: 1999:159764 USPATFULL
TITLE: ***Antisense*** modulation of microtubule-

INVENTOR(S): Bennett, C. Frank, Carlsbad, CA, United States
Ackermann, Elizabeth J., Solana Beach, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5998148		19991207	<--
APPLICATION INFO.:	US 1999-289368		19990408	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliot, George C.
ASSISTANT EXAMINER: Wang, Andrew
LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
LINE COUNT: 3094
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB ***Antisense*** compounds, compositions and methods are provided for modulating the expression of microtubule-associated protein 4. The compositions comprise ***antisense*** compounds, particularly ***antisense*** oligonucleotides, targeted to nucleic acids encoding microtubule-associated protein 4. Methods of using these compounds for modulation of microtubule-associated protein 4 expression and for treatment of diseases associated with expression of microtubule-associated protein 4 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 17 OF 37 USPATFULL
ACCESSION NUMBER: 1999:151195 USPATFULL
TITLE: GATA-6 transcription factor: compositions and methods
INVENTOR(S): Walsh, Kenneth, Carlisle, MA, United States
PATENT ASSIGNEE(S): St. Elizabeth's Medical Center, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5990092		19991123	<--
APPLICATION INFO.:	US 1997-927394		19970827	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degen, Nancy
ASSISTANT EXAMINER: Schwartzman, Robert
LEGAL REPRESENTATIVE: Wolf, Greenfield & Sacks P.C.
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 2449
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions for reducing or preventing the proliferation of vascular smooth muscle cells are provided. The method involves the step of administering an isolated GATA-6 molecule to a subject to prevent or reduce vascular smooth muscle cell proliferation. The isolated GATA-6 molecule can be a GATA-6 nucleic acid or a GATA-6 protein.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 18 OF 37 USPATFULL
ACCESSION NUMBER: 1999:96216 USPATFULL
TITLE: Reagents and methods useful for detecting diseases of the lung
INVENTOR(S): Cohen, Maurice, Highland Park, IL, United States
Friedman, Paula N., Deerfield, IL, United States
Gordon, Julian, Lake Bluff, IL, United States
Hodges, Steven C., Buffalo Grove, IL, United States
Klass, Michael R., Libertyville, IL, United States
Kratochvil, Jon D., Kenosha, WI, United States
Roberts-Rapp, Lisa, Gurnee, IL, United States
Russell, John C., Kenosha, WI, United States
Stroupe, Steven D., Libertyville, IL, United States
PATENT ASSIGNEE(S): Abbott Laboratories, Abbott Park, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE

APPLICATION INFO.: US 1997-964725 19971105 (8)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-744211, filed on 5 Nov 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degren, Nancy
ASSISTANT EXAMINER: Wang, Andrew
LEGAL REPRESENTATIVE: Becker, Cheryl L., Goller, Mimi C.
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 3052
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A set of contiguous and partially overlapping RNA sequences and polypeptides encoded thereby, designated as LU103 and transcribed from lung tissue is described. A fully sequenced clone representing the longest continuous sequence of LU103 is also disclosed. These sequences are useful for detecting, diagnosing, staging, monitoring, prognosticating, preventing or treating, or determining the predisposition of an individual to diseases and conditions of the lung such as lung cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 19 OF 37 USPATFULL
ACCESSION NUMBER: 1999:50839 USPATFULL
TITLE: Oligoribonucleotides for cleaving RNA
INVENTOR(S): Crooke, Stanley T., Carlsbad, CA, United States
PATENT ASSIGNEE(S): ISIS Pharmaceuticals, Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5898031		19990427	<--
APPLICATION INFO.:	US 1996-659440		19960606 (8)	

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: LeGuyader, John L.
LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz Mackiewicz & Norris LLP
NUMBER OF CLAIMS: 66
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 3150
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Oligomeric compounds including oligoribonucleotides and oligoribonucleosides are provided that have subsequences of 2-pentoribofuranosyl nucleosides that activate dsRNase. The oligoribonucleotides and oligoribonucleosides can include substituent groups for increasing binding affinity to complementary nucleic acid strand as well as substituent groups for increasing nuclease resistance. The oligomeric compounds are useful for diagnostics and other research purposes, for modulating the expression of a protein in organisms, and for the diagnosis, detection and treatment of other conditions susceptible to oligonucleotide therapeutics.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 20 OF 37 USPATFULL
ACCESSION NUMBER: 1999:24783 USPATFULL
TITLE: Therapeutic oligonucleotides targeting the human MDR1 and MRP genes
INVENTOR(S): Smith, Larry J., Omaha, NE, United States
PATENT ASSIGNEE(S): The Board of Regents of the University of Nebraska, Lincoln, NE, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5874567		19990223	<--
APPLICATION INFO.:	US 1997-927561		19970908 (8)	

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-487141, filed on 7 Jun 1995, now patented, Pat. No. US 5683987 which is a continuation-in-part of Ser. No. US 1994-379180, filed on 12 Jul 1994, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: LeGuyader, John L.

LEGAL REPRESENTATIVE: Dann, Dorfman, Herrell and Skillman
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 2080
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel compositions and methods useful in cancer therapy for inhibiting the multidrug resistance phenotype, which often thwarts long-term chemotherapeutic regimens. The novel compositions of matter comprise oligonucleotides targeted to the human MDR1 and MRP genes, which inhibit expression of these genes, thereby rendering tumors and other forms of cancer more susceptible to the cytotoxic effects of chemotherapeutic agents. Oligonucleotides are also provided that inhibit the multidrug resistance phenotype by exerting an aptameric effect.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 21 OF 37 USPATFULL
ACCESSION NUMBER: 1999:18912 USPATFULL
TITLE: Method of determining DNA sequence preference of a DNA-binding molecule
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
Turin, Lisa M., Redwood City, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5869241		19990209	<--
APPLICATION INFO.:	US 1995-475228		19950607 (8)	

RELATED APPLN. INFO.: Division of Ser. No. US 1993-171389, filed on 20 Dec 1993, now patented, Pat. No. US 5578444 which is a continuation-in-part of Ser. No. US 1993-123936, filed on 17 Sep 1993, now patented, Pat. No. US 5726014 which is a continuation-in-part of Ser. No. US 1992-996783, filed on 23 Dec 1992, now patented, Pat. No. US 5693463 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Zitomer, Stepanie W.
ASSISTANT EXAMINER: Whisenant, Ethan
LEGAL REPRESENTATIVE: Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 72 Drawing Figure(s); 47 Drawing Page(s)
LINE COUNT: 9840
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines a DNA:protein-binding assay useful for screening libraries of synthetic or biological compounds for their ability to bind DNA test sequences. The assay is versatile in that any number of test sequences can be tested by placing the test sequence adjacent to a defined protein binding screening sequence. Binding of molecules to these test sequence changes the binding characteristics of the protein molecule to its cognate binding sequence. When such a molecule binds the test sequence the equilibrium of the DNA:protein complexes is disturbed, generating changes in the concentration of free DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ ID NO:600) are set forth. The assay of the present invention is also useful to characterize the preferred binding sequences of any selected DNA-binding molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 22 OF 37 USPATFULL
ACCESSION NUMBER: 1999:4647 USPATFULL
TITLE: Fas ligand compositions for treatment of proliferative disorders
INVENTOR(S): Walsh, Kenneth, Carlisle, MA, United States
PATENT ASSIGNEE(S): St. Elizabeth's Medical Center, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5858990		19990112	<--
APPLICATION INFO.:	US 1997-810453		19970304	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Wolf, Greenfield & Sacks, P.C.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 3038
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for treating vascular injury, particularly vascular injury resulting from restenosis following angioplasty, and vascular remodeling is provided. The method involves administering to subjects in need of such treatment an effective amount of a Fas ligand molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 23 OF 37 USPATFULL
ACCESSION NUMBER: 1998:151104 USPATFULL
TITLE: Modified oligonucleotides, their preparation and their use
INVENTOR(S): Seela, Frank, Osnabrueck, Germany, Federal Republic of
Thomas, Horst, Hasbergen, Germany, Federal Republic of
PATENT ASSIGNEE(S): Hoechst Aktiengesellschaft, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5844106		19981201	<--
APPLICATION INFO.:	US 1995-554164		19951106	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Marschel, Ardin H.
ASSISTANT EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Foley & Lardner
NUMBER OF CLAIMS: 17
EXEMPLARY CLAIM: 1
LINE COUNT: 2731
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Modified oligonucleotides which possess at least one substituted 7-deazapurine base form more stable hybridization complexes with nucleic acids than unsubstituted analogs. They are useful as inhibitors of gene expression, as probes for detecting nucleic acids, as aids in molecular biology and as pharmaceuticals or diagnostic agents. Processes for preparing

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 24 OF 37 USPATFULL
ACCESSION NUMBER: 1998:150686 USPATFULL
TITLE: Cleavage of nucleic acid acid using thermostable methoanococcus jannaschii FEN-1 endonucleases
INVENTOR(S): Kaiser, Michael W., Madison, WI, United States
Lyamichev, Victor I., Madison, WI, United States
Lyamichev, Natasha, Madison, WI, United States
PATENT ASSIGNEE(S): Third Wave Technologies, Inc., Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5843669		19981201	<--
APPLICATION INFO.:	US 1996-757653		19961129	(8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-599491, filed on 24 Jan 1996
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Medlen & Carroll, LLP
NUMBER OF CLAIMS: 26
EXEMPLARY CLAIM: 3
NUMBER OF DRAWINGS: 161 Drawing Figure(s); 131 Drawing Page(s)
LINE COUNT: 15189

AB The present invention relates to means for cleaving a nucleic acid cleavage structure in a site-specific manner. Structure-specific nucleases, including 5' nucleases, thermostable FEN-1 endonucleases and 3' exonucleases, are used to detect and identify target nucleic acids. Methods are provided which allow for the detection specific nucleic acid sequences; these methods permit the detection and identification of mutant and wild-type forms of genes (e.g., human genes) as well as permit the detection and identification of bacterial and viral pathogens in a sample.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 25 OF 37 USPATFULL
ACCESSION NUMBER: 1998:150671 USPATFULL
TITLE: Rapid detection of mutations in the ***p53*** gene
INVENTOR(S): Heisler, Laura M., Madison, WI, United States
Fors, Lance, Monrovia, CA, United States
Brow, Mary Ann D., Madison, WI, United States
PATENT ASSIGNEE(S): Third Wave Technologies, Inc., Madison, WI, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5843654		19981201	<--
APPLICATION INFO.:	US 1995-484956		19950607	(8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-402601, filed on 9 Mar 1995 which is a continuation-in-part of Ser. No. US 1994-337164, filed on 9 Nov 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-254359, filed on 6 Jun 1994, now patented, Pat. No. US 5614402, issued on 25 Mar 1997 which is a continuation-in-part of Ser. No. US 1993-73384, filed on 4 Jun 1993, now patented, Pat. No. US 5541311, issued on 30 Jul 1996 which is a continuation-in-part of Ser. No. US 1992-986330, filed on 7 Dec 1992, now patented, Pat. No. US 5422253, issued on 6 Jun 1995
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Patterson, Jr., Charles L.
LEGAL REPRESENTATIVE: Medlen & Carroll, LLP
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 118 Drawing Figure(s); 79 Drawing Page(s)
LINE COUNT: 11066
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to means for cleaving a nucleic acid cleavage structure in a site-specific manner. Enzymes, including 5' nucleases and 3' exonucleases, are used to screen for known and unknown mutations, including single base changes, in the human ***p53*** gene. Methods are provided which allow for the identification of genetic mutations in the human ***p53*** gene in a sample.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 26 OF 37 USPATFULL
ACCESSION NUMBER: 1998:92178 USPATFULL
TITLE: Nucleotide monomers containing 8-azapurin bases or a derivative thereof, their preparation and their use in making modified olignonucleotides
INVENTOR(S): Seela, Frank, Osnabruck, Germany, Federal Republic of
Lampe, Sigrid, Berge/Hekese, Germany, Federal Republic of
PATENT ASSIGNEE(S): Hoechst Aktiengesellschaft, Frankfurt am Main, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5789562		19980804	<--
APPLICATION INFO.:	US 1997-940196		19970929	(8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-431777, filed on 1 May 1995, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	DE 1994-4415370	19940502

DOCUMENT TYPE: Utility

PRIMARY EXAMINER: Marschel, Ardin H.
ASSISTANT EXAMINER: Riley, Jezia
LEGAL REPRESENTATIVE: Finnegan, Henderson, Farabow, Garrett & Dunner, L.L.P.
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
LINE COUNT: 1868
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to novel modified oligonucleotides which contain at least one 8-azapurine base and form more stable hybridization complexes with nucleic acids; To a process for their preparation, and to their use as inhibitors of gene expression, as probes for detecting nucleic acids, as aids in molecular biology, and as a pharmaceutical or diagnostic agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 27 OF 37 USPATFULL
ACCESSION NUMBER: 1998:68824 USPATFULL
TITLE: Targeted nucleic acid delivery into liver cells
INVENTOR(S): Kuo, M. Tien, Houston, TX, United States
Ding, Zhi-Ming, Houston, TX, United States
PATENT ASSIGNEE(S): Board of Regents , The University of Texas System, Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5766899		19980616	<--
APPLICATION INFO.:	US 1995-395602		19950227	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Robinson, Douglas W.
ASSISTANT EXAMINER: Nelson, Amy J.
LEGAL REPRESENTATIVE: Arnold, White & Durkee
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 26
NUMBER OF DRAWINGS: 20 Drawing Figure(s); 15 Drawing Page(s)
LINE COUNT: 1842
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a receptor-mediated complex that selectively delivers nucleic acid into hepatocytes. Circumsporozoite (CS) protein is the targeting ligand that recognizes a receptor expressed on the liver cell surface. The CS ligand is complexed with a polylysine component that can bind nucleic acid. The level of gene expression is greatly enhanced when the complex is cotransfected with adenovirus. Using the present invention, a reporter gene was successfully transferred into a number of different cell lines that express high levels of receptor. The ability to introduce nucleic acid into specific mammalian cells is an important therapy for numerous diseases such as cancer, malaria and hepatitis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 28 OF 37 USPATFULL
ACCESSION NUMBER: 1998:44877 USPATFULL
TITLE: Sequence-directed DNA-binding molecules compositions and methods
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5744131		19980428	<--
APPLICATION INFO.:	US 1995-476876		19950607	(8)

RELATED APPLN. INFO.: Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Zitomer, Stephanie W.
ASSISTANT EXAMINER: Atzel, Amy
LEGAL REPRESENTATIVE: Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 3

NUMBER OF DRAWINGS: 48 Drawing Figure(s); 33 Drawing Page(s)
LINE COUNT: 5113
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific DNA test sequences. The assay is also useful for determining the sequence specificity and relative DNA-binding affinity of DNA-binding molecules for any particular DNA sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific DNA-binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 29 OF 37 USPATFULL
ACCESSION NUMBER: 1998:39383 USPATFULL
TITLE: Sequence-directed DNA-binding molecules compositions and methods
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5738990		19980414	<--
APPLICATION INFO.:	US 1995-475221		19950607 (8)	

RELATED APPLN. INFO.: Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Guzo, David
ASSISTANT EXAMINER: Brusca, John S.
LEGAL REPRESENTATIVE: Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Figure(s); 33 Drawing Page(s)
LINE COUNT: 5040
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific DNA test sequences. The assay is also useful for determining the sequence specificity and relative DNA-binding affinity of DNA-binding molecules for any particular DNA sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific DNA-binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 30 OF 37 USPATFULL
ACCESSION NUMBER: 1998:31048 USPATFULL
TITLE: Method of use of radicicol for treatment of immunopathological disorders
INVENTOR(S): Feng, Lili, San Diego, CA, United States
Hwang, Daniel, Baton Rouge, LA, United States
PATENT ASSIGNEE(S): The Scripps Research Institute, La Jolla, CA, United States (U.S. corporation)
Board of Supervisors of Louisiana State University and Agricultural and Mechanical College, Baton Rouge, LA,

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5731343		19980324	<--
APPLICATION INFO.:	US 1995-394148		19950224	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Cintins, Marianne M.
ASSISTANT EXAMINER: Jarvis, William R. A.
LEGAL REPRESENTATIVE: Fish & Richardson, P.C.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT: 1540
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a method of treating an immunopathological disorder having an etiology associated with production of a proinflammatory agent, by administering a compound of the formula: ##STR1## where R1 and R2 are independently H or --COR3; R3 is H, 1-50C alkyl, 1-20C alkoxy, 2-30C alkenyl, 2-30C alkenyloxy, 2-10 alkynyl, 6-14C aryl or aryloxy, a 5-6 membered heterocycle (containing 1-3 N, O and/or S heteroatoms and optionally fused to an aryl group), 3-8C cycloalkyl (optionally fused to aryl) or 5-8C cycloalkenyl; and R4 is a halogen. Examples of such proinflammatory agents include interleukin-1 (IL-1), interleukin-6 (IL-6), interferon-.gamma. (IFN-.gamma.), tumor necrosis factor-.alpha. (TNF-.alpha.), granulocyte macrophage-colony stimulating factor (GM-CSF), the growth related gene KC, cyclooxygenase-1 (COX-1), cyclooxygenase-2 (COX-2), macrophage chemotactic protein (MCP), inducible nitric oxide synthetase (iNOS), macrophage inflammatory protein (MIP), tissue factor (TF), phosphotyrosine phosphatase (PTPase), and endotoxin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 31 OF 37 USPATFULL
ACCESSION NUMBER: 1998:25075 USPATFULL
TITLE: Screening assay for the detection of DNA-binding molecules
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Watertown, MA, United States
Turin, Lisa M., Berkeley, CA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5726014		19980310	<--
APPLICATION INFO.:	US 1993-123936		19930917	(8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Atzel, Amy
LEGAL REPRESENTATIVE: Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 72 Drawing Figure(s); 47 Drawing Page(s)
LINE COUNT: 5659
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention defines a DNA:protein-binding assay useful for screening libraries of synthetic or biological compounds for their ability to bind DNA test sequences. The assay is versatile in that any number of test sequences can be tested by placing the test sequence adjacent to a defined protein binding screening sequence. Binding of molecules to these test sequence changes the binding characteristics of the protein molecule to its cognate binding sequence. When such a molecule binds the test sequence the equilibrium of the DNA:protein complexes is disturbed, generating changes in the concentration of free DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ ID NO:600) are set forth. The assay of the present invention is also useful to characterize the preferred binding sequences of any selected DNA-binding molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 32 OF 37 USPATFULL
ACCESSION NUMBER: 1998:14634 USPATFULL
TITLE: Method of constructing sequence-specific DNA-binding molecules
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Watertown, MA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5716780		19980210	<--
APPLICATION INFO.:	US 1995-484499		19950607 (8)	

RELATED APPLN. INFO.: Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Atzel, Amy
LEGAL REPRESENTATIVE: Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 48 Drawing Figure(s); 33 Drawing Page(s)
LINE COUNT: 4929
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific DNA test sequences. The assay is also useful for determining the sequence specificity and relative DNA-binding affinity of DNA-binding molecules for any particular DNA sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific DNA-binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 33 OF 37 USPATFULL
ACCESSION NUMBER: 1998:11870 USPATFULL
TITLE: Rolling circle synthesis of oligonucleotides and amplification of select randomized circular oligonucleotides
INVENTOR(S): Kool, Eric T., Rochester, NY, United States
PATENT ASSIGNEE(S): University of Rochester, Rochester, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5714320		19980203	<--
APPLICATION INFO.:	US 1995-393439		19950223 (8)	

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-47860, filed on 15 Apr 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Rees, Dianne
LEGAL REPRESENTATIVE: Mueting, Raasch, Gebhardt & Schwappach, P.A.
NUMBER OF CLAIMS: 47
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 2583
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods for synthesis, selection, and amplification of DNA and RNA oligonucleotides and analogs. The method for synthesizing an oligonucleotide involves: providing an effective

at least one copy of the desired oligonucleotide sequence linked to a cleavage site; providing an effective amount of an isolated oligonucleotide primer; annealing the primer to the circular template to form a primed circular template; and combining the primed circular template with an effective amount of at least two types of nucleotide triphosphates and an effective amount of a polymerase enzyme to form a nucleotide multimer complementary to the circular oligonucleotide template, wherein the nucleotide multimer comprises multiple copies of the oligonucleotide sequence joined end to end. Preferably, the nucleotide multimer is cleaved to produce oligonucleotides having well-defined ends.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 34 OF 37 USPATFULL

ACCESSION NUMBER:	97:112300 USPATFULL			
TITLE:	Method of ordering sequence binding preferences of a DNA-binding molecule			
INVENTOR(S):	Edwards, Cynthia A., Menlo Park, CA, United States Fry, Kirk E., Palo Alto, CA, United States Cantor, Charles R., Boston, MA, United States Andrews, Beth M., Maynard, MA, United States(4)			
PATENT ASSIGNEE(S):	Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5693463		19971202	<--
APPLICATION INFO.:	US 1992-996783		19921223	(7)
DISCLAIMER DATE:	20110426			
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Zitomer, Stephanie W.			
ASSISTANT EXAMINER:	Atzel, Amy			
LEGAL REPRESENTATIVE:	Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.			
NUMBER OF CLAIMS:	3			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	48 Drawing Figure(s); 33 Drawing Page(s)			
LINE COUNT:	4908			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific DNA test sequences. The assay is also useful for determining the sequence specificity and relative DNA-binding affinity of DNA-binding molecules for any particular DNA sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific DNA-binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 35 OF 37 USPATFULL

ACCESSION NUMBER:	97:101743 USPATFULL			
TITLE:	Therapeutic oligonucleotides targeting the human MDR1 and MRP genes			
INVENTOR(S):	Smith, Larry J., Omaha, NE, United States			
PATENT ASSIGNEE(S):	The Board of Regents of the University of Nebraska, Lincoln, NE, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5683987		19971104	<--
APPLICATION INFO.:	US 1995-487141		19950607	(8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1994-379180, filed on 12 Jul 1994, now abandoned			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			

ASSISTANT EXAMINER: Wang, Andrew
LEGAL REPRESENTATIVE: Dann, Dorfman, Herrell and Skillman
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 2111
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel compositions and methods useful in cancer therapy for inhibiting the multidrug resistance phenotype, which often thwarts long-term chemotherapeutic regimens. The novel compositions of matter comprise oligonucleotides targeted to the human MDR1 and MRP genes, which inhibit expression of these genes, thereby rendering tumors and other forms of cancer more susceptible to the cytotoxic effects of chemotherapeutic agents. Oligonucleotides are also provided that inhibit the multidrug resistance phenotype by exerting an aptameric effect.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 36 OF 37 USPATFULL
ACCESSION NUMBER: 97:17918 USPATFULL
TITLE: Compositions and methods for enhanced drug delivery
INVENTOR(S): Hale, Ron L., Woodside, CA, United States
Lu, Amy, Los Altos, CA, United States
Solas, Dennis, San Francisco, CA, United States
Selick, Harold E., Belmont, CA, United States
Oldenburg, Kevin R., Fremont, CA, United States
Zaffaroni, Alejandro C., Atherton, CA, United States
PATENT ASSIGNEE(S): Affymax Technologies N.V., Middlesex, England (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5607691		19970304	<--
APPLICATION INFO.:	US 1995-449188		19950524 (8)	

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-164293, filed on 9 Dec 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-77296, filed on 14 Jun 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-898219, filed on 12 Jun 1992, now abandoned And a continuation-in-part of Ser. No. US 1993-9463, filed on 27 Jan 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Levy, Neil S.
LEGAL REPRESENTATIVE: Stevens, Lauren L.
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
LINE COUNT: 5349
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods of delivering pharmaceutical agents across membranes, including the skin layer or mucosal membranes of a patient. A pharmaceutical agent is covalently bonded to a chemical modifier, via a physiologically cleavable bond, such that the membrane transport and delivery of the agent is enhanced.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 37 OF 37 USPATFULL
ACCESSION NUMBER: 96:108816 USPATFULL
TITLE: Sequence-directed DNA-binding molecules compositions and methods
INVENTOR(S): Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
Turin, Lisa M., Redwood City, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
PATENT ASSIGNEE(S): Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5578444		19961126	<--
APPLICATION INFO.:	US 1993-171389		19931220 (8)	

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-123936, filed on 17 Sep 1993 which is a continuation-in-part of Ser.

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                        continuation-in-part of Ser. No. US 1991-723618, filed
                        on 27 Jun 1991, now abandoned
DOCUMENT TYPE:          Utility
FILE SEGMENT:           Granted
PRIMARY EXAMINER:       Zitomer, Stephanie W.
ASSISTANT EXAMINER:     Atzel, Amy
LEGAL REPRESENTATIVE:   Fabian, Gary R., Brookes, Allen A., Stratford, Carol A.
NUMBER OF CLAIMS:       15
EXEMPLARY CLAIM:        1
NUMBER OF DRAWINGS:     71 Drawing Figure(s); 48 Drawing Page(s)
LINE COUNT:             5845
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB      The present invention defines a DNA:protein-binding assay useful for
        screening libraries of synthetic or biological compounds for their
        ability to bind DNA test sequences. The assay is versatile in that any
        number of test sequences can be tested by placing the test sequence
        adjacent to a defined protein binding screening sequence. Binding of
        molecules to these test sequence changes the binding characteristics of
        the protein molecule to its cognate binding sequence. When such a
        molecule binds the test sequence the equilibrium of the DNA:protein
        complexes is disturbed, generating changes in the concentration of free
        DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ
        ID NO:600) are set forth. The assay of the present invention is also
        useful to characterize the preferred binding sequences of any selected
        DNA-binding molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> d history

   (FILE 'HOME' ENTERED AT 19:01:02 ON 21 JAN 2003)

     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     19:01:24 ON 21 JAN 2003
L1          4478 S P53 AND ANTISENSE
L2            78 S L1 AND SPLICE ACCEPTOR SITE
L3            78 DUP REM L2 (0 DUPLICATES REMOVED)
L4             2 S L3 AND MORPHOLINO
L5           171 S L1 AND MORPHOLINO
L6           157 DUP REM L5 (14 DUPLICATES REMOVED)
L7            37 S L6 AND PY<2001

=> s p53 (p) antisense
L8          2189 P53 (P) ANTISENSE

=> s l8 and morpholino
L9            66 L8 AND MORPHOLINO

=> s l9 not l7
L10           54 L9 NOT L7

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11           41 DUP REM L10 (13 DUPLICATES REMOVED)

=> d l11 ibib abs tot

L11  ANSWER 1 OF 41  USPATFULL
ACCESSION NUMBER:       2003:17392  USPATFULL
TITLE:                  HS2STs as modifiers of the p53 pathway and methods of
                        use
INVENTOR(S):            Friedman, Lori, San Francisco, CA, UNITED STATES
                        Plowman, Gregory D., San Carlos, CA, UNITED STATES
                        Belvin, Marcia, Albany, CA, UNITED STATES
                        Francis-Lang, Helen, San Francisco, CA, UNITED STATES
                        Li, Danxi, San Francisco, CA, UNITED STATES
                        Funke, Roel P., South San Francisco, CA, UNITED STATES

                             NUMBER         KIND    DATE
                        ---------------- ------ --------
PATENT INFORMATION:     US 2003013144      A1   20030116
APPLICATION INFO.:      US 2002-161398     A1   20020603  (10)

                               NUMBER         DATE
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US 2001-328605P 20011010 (60)
US 2002-357253P 20020215 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JAN P. BRUNELLE, EXELIXIS, INC., 170 HARBOR WAY, P.O. BOX 511, SOUTH SAN FRANCISCO, CA, 94083-0511
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
LINE COUNT: 2023
AB Human HS2ST genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders associated with defective p53 function. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of HS2ST are provided.

L11 ANSWER 2 OF 41 USPATFULL DUPLICATE 1
ACCESSION NUMBER: 2002:69973 USPATFULL
TITLE: ***p53*** ***antisense*** agent and method
INVENTOR(S): Iversen, Patrick L., Corvallis, OR, United States
PATENT ASSIGNEE(S): AVI BioPharma, Inc., Corvallis, OR, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6365577	B1	20020402
APPLICATION INFO.:	US 1999-426804		19991022 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-105695P	19981026 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Wang, Andrew
ASSISTANT EXAMINER: Zara, Jane
LEGAL REPRESENTATIVE: Gorthey, LeeAnn
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 1006
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB ***Antisense*** oligonucleotides useful for treating a disease state characterized by ***p53*** induction, such as proliferative cell disorders, e.g. cancer, or a hypoxic state induced by an ischemic attack, such as stroke, are described. The ***antisense*** agents are preferably of the class known as "steric blocker" type oligonucleotides, including ***morpholino*** oligonucleotides, peptide nucleic acids, 2'-O-allyl or 2'-O-alkyl modified oligonucleotides, or N3'.fwdarw.P5' phosphoramidate oligonucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 3 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946579 CAPLUS
DOCUMENT NUMBER: 138:21196
TITLE: Peroxisomal enoyl-CoA isomerases as modifiers of the p53 pathway and their use in diagnosis and treatment of p53-related diseases
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099426	A1	20021212	WO 2002-US17420	20020603

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.:
US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Two human peroxisomal .DELTA.3,.DELTA.2-enoyl-CoA isomerase (PECI) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The CG13890 gene was identified as a modifier of the p53 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, PECI genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of PECI are provided.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 4 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946511 CAPLUS
DOCUMENT NUMBER: 138:21195
TITLE: Glycine receptor chloride channel proteins as modifiers of the p53 pathway and their use in diagnosis and treatment of p53-related diseases
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099140	A1	20021212	WO 2002-US17458	20020602

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.:
US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Three human glycine receptor chloride channel .alpha.-subunit (GLRA) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The CG14723 gene was identified as a modifier of the p53 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, GLRA genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of GLRA are provided.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946509 CAPLUS
DOCUMENT NUMBER: 138:21194
TITLE: Human sulfotransferase proteins as modifiers of the p53 pathway and their use in diagnosis and treatment of p53-related diseases

Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099138	A2	20021212	WO 2002-US17409	20020603
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.: US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Four human sulfotransferase proteins with transferase domains (HS2ST) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The pipe gene was identified as a modifier of the p53 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, HS2ST genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of HS2ST are provided.

L11 ANSWER 6 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946461 CAPLUS
DOCUMENT NUMBER: 138:35033
TITLE: cDNA and protein sequences of human glutamine fructose-6-phosphate amidotransferase and the uses of the protein as modifiers of the p53 pathway in diagnosis and therapeutics
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099083	A2	20021212	WO 2002-US21112	20020602
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.: US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Human glutamine fructose-6-phosphate amidotransferase (GFAT) genes are identified as modulators of the p53 pathway, and thus are therapeutic

screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The human GFAT gene was identified by Blast searching of mouse GFAT counterparts and the invention also provides distribution pattern of human GFAT gene in normal and tumor tissue cells. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, GFAT genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of GFAT are provided.

L11 ANSWER 7 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946453 CAPLUS
DOCUMENT NUMBER: 138:35032
TITLE: Protein arginine N-methyltransferase as modifiers of the p53 pathway and their use in diagnosis and treatment of p53-related diseases
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 67 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099075	A2	20021212	WO 2002-US17879	20020605

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2001-338733P P 20011022
US 2002-357253P P 20020215
US 2002-357600P P 20020215

AB Human and mouse protein arginine N-methyltransferase (PRMT) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The CG5358 gene was identified as a modifier of the p53 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, PRMT genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of PRMT are provided.

L11 ANSWER 8 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946452 CAPLUS
DOCUMENT NUMBER: 138:34194
TITLE: cDNA and protein sequences of human amino acid transporter SLC7s and the uses of the protein as modifiers of the p53 pathway in diagnosis and therapeutics
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 129 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2001-338733P P 20011022
US 2002-357253P P 20020215
US 2002-357600P P 20020215

AB Human amino acid transporter SLC7 genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The human SLC7 genes were identified by Blast searching of mouse SLC7 counterparts and the invention also provides distribution pattern of human SLC7 genes in normal and tumor tissue cells. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, SLC7 genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of SLC7 are provided.

L11 ANSWER 9 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946437 CAPLUS
DOCUMENT NUMBER: 138:20565
TITLE: cDNA and protein sequences of human U5-snRNP-specific protein U5-200KD and the uses of the protein as modifiers of the p53 pathway in diagnosis and therapeutics
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099059	A2	20021212	WO 2002-US17524	20020603

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.: US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Human U5-snRNP-specific protein U5-200KD genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The human U5-200KD genes were identified by Blast searching of mouse U5-200KD counterparts and the invention also provides distribution pattern of human U5-200KD genes in normal and tumor tissue cells. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, U5-200KD genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that

L11 ANSWER 10 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946436 CAPLUS
DOCUMENT NUMBER: 138:34193
TITLE: cDNA and protein sequences of human potassium channel and the uses of the protein as modifiers of the p53 pathway in diagnosis and therapeutics
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 62 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099058	A2	20021212	WO 2002-US17476	20020603
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002192695	A1	20021219	US 2002-161510	20020603
US 2003013144	A1	20030116	US 2002-161398	20020603

PRIORITY APPLN. INFO.: US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Human potassium large conductance calcium-activated channel (subfamily M) alpha member (KCNMA) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The human KCNMA genes were identified by Blast searching of mouse KCNMA counterparts and the invention also provides distribution pattern of human KCNMA genes in normal and tumor tissue cells. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, KCNMA genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of KCNMA are provided.

L11 ANSWER 11 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946435 CAPLUS
DOCUMENT NUMBER: 138:21191
TITLE: RNA-binding KH domain-containing proteins as modifiers of the p53 pathway and their use in diagnosis and treatment of p53-related diseases
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099057	A2	20021212	WO 2002-US17475	20020603
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002192695	A1	20021219	US 2002-161510		20020603
US 2003013144	A1	20030116	US 2002-161398		20020603

PRIORITY APPLN. INFO.:
US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Four human RNA-binding proteins with KH domains (SAM) genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The qkr58E-2 gene was identified as a modifier of the p53 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, SAM genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of SAM are provided.

L11 ANSWER 12 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:946434 CAPLUS
DOCUMENT NUMBER: 138:20564
TITLE: cDNA and protein sequences of human protein C22C7ORF and the uses of the protein as modifiers of the p53 pathway in diagnosis and therapeutics
INVENTOR(S): Friedman, Lori; Plowman, Gregory D.; Belvin, Marcia; Francis-Lang, Helen; Li, Danxi; Funke, Roel P.
PATENT ASSIGNEE(S): Exelixis, Inc., USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 37
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002099056	A2	20021212	WO 2002-US17474	20020603

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2002192695	A1	20021219	US 2002-161510		20020603
US 2003013144	A1	20030116	US 2002-161398		20020603

PRIORITY APPLN. INFO.:
US 2001-296076P P 20010605
US 2001-328605P P 20011010
US 2002-357253P P 20020215

AB Human protein C22C7ORF genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders assocd. with defective p53 function. Genetic screens were designed to identify modifiers of the p53 pathway in Drosophila in which p53 was overexpressed in the wing. The human protein C22C7ORF genes were identified by Blast searching of mouse protein C22C7ORF counterparts and the invention also provides distribution pattern of human protein C22C7ORF genes in normal and cancerous tissue cells. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, protein C22C7ORF genes and proteins are attractive drug targets for the treatment of pathologies assocd. with a defective p53 signaling pathway, such as cancer. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of protein C22C7ORF are provided.

L11 ANSWER 13 OF 41 USPATFULL
ACCESSION NUMBER: 2002:337337 USPATFULL
TITLE: PIBs as modifiers of the p53 pathway and methods of use
INVENTOR(S): Friedman, Lori, San Francisco, CA, UNITED STATES
Plowman, Gregory D., San Carlos, CA, UNITED STATES
Belvin, Marcia, Albany, CA, UNITED STATES
Francis-Lang, Helen, San Francisco, CA, UNITED STATES
Li, Danxi, San Francisco, CA, UNITED STATES
Funke, Roel P., South San Francisco, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002192695	A1	20021219
APPLICATION INFO.:	US 2002-161510	A1	20020603 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-296076P	20010605 (60)
	US 2001-328605P	20011010 (60)
	US 2002-357253P	20020215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JAN P. BRUNELLE, EXELIXIS, INC., 170 HARBOR WAY, P.O. BOX 511, SOUTH SAN FRANCISCO, CA, 94083-0511
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
LINE COUNT: 4841
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Human PIB genes are identified as modulators of the p53 pathway, and thus are therapeutic targets for disorders associated with defective p53 function. Methods for identifying modulators of p53, comprising screening for agents that modulate the activity of PIB are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 14 OF 41 USPATFULL
ACCESSION NUMBER: 2002:301576 USPATFULL
TITLE: Inhibition of ATF2 activity to treat cancer
INVENTOR(S): Ronai, Ze?apos,ev, Suffern, NY, UNITED STATES
PATENT ASSIGNEE(S): Mount Sinai School of Medicine (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002169121	A1	20021114
APPLICATION INFO.:	US 2002-76905	A1	20020214 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-269257P	20010216 (60)
	US 2001-269118P	20010215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: DARBY & DARBY P.C., 805 Third Avenue, New York, NY, 10022
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 3146

AB The present invention relates to novel therapies for cancer and, in particular, to therapies that are particularly suited to tumor cells resistant to other types of therapies such as radiation, chemotherapy, or combinations of both approaches. The invention provides methods for identifying and implementing strategies to inhibit a transcription factor which, in combination with other factors, renders the cells resistant and inhibits apopotosis of the cells. The invention provides an inhibitory ATF2 N-terminal fragment, specifically a fragment corresponding to amino acid residues 50-100 of ATF2 (termed peptide II). The invention provides methods for inhibiting tumor cell growth with such peptides.

L11 ANSWER 15 OF 41 USPATFULL
ACCESSION NUMBER: 2002:295143 USPATFULL
TITLE: Oligoribonucleotides and ribonucleases for cleaving RNA
INVENTOR(S): Crooke, Stanley T., Carlsbad, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002165189	A1	20021107
APPLICATION INFO.:	US 2002-78949	A1	20020220 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-479783, filed on 7 Jan 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Woodcock Washburn LLP, One Liberty Place, 46th Floor, Philadelphia, PA, 19103

EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 3922
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Oligomeric compounds including oligoribonucleotides and oligoribonucleosides are provided that have subsequences of 2'-pentoribofuranosyl nucleosides that activate dsRNase. The oligoribonucleotides and oligoribonucleosides can include substituent groups for increasing binding affinity to complementary nucleic acid strand as well as substituent groups for increasing nuclease resistance. The oligomeric compounds are useful for diagnostics and other research purposes, for modulating the expression of a protein in organisms, and for the diagnosis, detection and treatment of other conditions susceptible to oligonucleotide therapeutics. Also included in the invention are mammalian ribonucleases, i.e., enzymes that degrade RNA, and substrates for such ribonucleases. Such a ribonuclease is referred to herein as a dsRNase, wherein "ds" indicates the RNase's specificity for certain double-stranded RNA substrates. The artificial substrates for the dsRNases described herein are useful in preparing affinity matrices for purifying mammalian ribonuclease as well as non-degradative RNA-binding proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 16 OF 41 USPATFULL
ACCESSION NUMBER: 2002:294546 USPATFULL
TITLE: Detection of nucleic acid heteroduplex molecules by anion-exchange chromatography
INVENTOR(S): Taylor, Paul D., Gilroy, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002164589	A1	20021107
APPLICATION INFO.:	US 2001-756070	A1	20010106 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-687834, filed on 11 Oct 2000, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-194652P	20000404 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JOHN F. BRADY, TRANSGENOMIC, INC., 2032 CONCOURSE DRIVE, SAN JOSE, CA, 95131
NUMBER OF CLAIMS: 72
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 2151
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention describes a method for separating or partially separating heteroduplex and homoduplex DNA molecules in a mixture. In the method, the mixture is applied to an anion-exchange chromatography medium. The heteroduplex and homoduplex molecules are eluted with a mobile phase containing an eluting salt, including an anion and a cation, a buffer, and preferably including an organic solvent. The eluting is carried out under conditions effective to at least partially denature the heteroduplexes (e.g., thermal or chemical denaturing) resulting in the separation of the heteroduplexes from the homoduplexes. The method has many applications including, but not limited to, detecting mutations and comparative DNA sequencing.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 17 OF 41 USPATFULL
ACCESSION NUMBER: 2002:273383 USPATFULL
TITLE: Antisense oligonucleotide modulation of human MDM2 expression
INVENTOR(S): Miraglia, Loren J., Encinitas, CA, UNITED STATES
Nero, Pamela, Oceanside, CA, UNITED STATES
Graham, Mark J., San Clemente, CA, UNITED STATES
Monia, Brett P., La Costa, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002151511	A1	20021017
APPLICATION INFO.:	US 2001-851771	A1	20010509 (9)

1998, GRANTED, Pat. No. US 6238921
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 E. Main Street, Marlton, NJ, 08053
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
LINE COUNT: 1409
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compounds, compositions and methods are provided for inhibiting the expression of human mdm2. The compositions comprise antisense oligonucleotides targeted to nucleic acids encoding mdm2. Methods of using these oligonucleotides for inhibition of mdm2 expression and for treatment of diseases such as cancers associated with overexpression of mdm2 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 18 OF 41 USPATFULL
ACCESSION NUMBER: 2002:272813 USPATFULL
TITLE: Diagnosis and treatment of cancer using mammalian pellino polypeptides and polynucleotides
INVENTOR(S): Powers, Scott, Greenlawn, NY, UNITED STATES
Mu, David, Jericho, NY, UNITED STATES
Xiang, Phil, San Francisco, CA, UNITED STATES
Peng, Yue, South Setauket, NY, UNITED STATES
PATENT ASSIGNEE(S): Tularik Inc., South San Francisco, CA, 94080 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002150934	A1	20021017
APPLICATION INFO.:	US 2001-41030	A1	20011228 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-259502P	20010102 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 37
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 3106
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods, reagents, and kits for diagnosing and treating cancer in a mammal, e.g., a human. This invention is based upon the discovery that Pellino 1 or 2 is overexpressed and/or amplified in cancer. Methods to detect cancer or a propensity to develop cancer, to monitor the efficacy of a cancer treatment, and to treat cancer, by inhibiting the expression and/or activity of Pellino 1 or 2 in a cancer cell are included.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 19 OF 41 USPATFULL
ACCESSION NUMBER: 2002:266324 USPATFULL
TITLE: Tyrosine kinase inhibitors
INVENTOR(S): Bilodeau, Mark T., Lansdale, PA, UNITED STATES
Hartman, George D., Lansdale, PA, UNITED STATES
Manley, Peter J., Harleysville, PA, UNITED STATES
PATENT ASSIGNEE(S): Merck & Co., Inc. (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002147203	A1	20021010
APPLICATION INFO.:	US 2002-62351	A1	20020201 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-658680, filed on 8 Sep 2000, ABANDONED

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-153348P	19990910 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

RY60-30, Rahway, NJ, 07065-0907
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 3989
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which contain these compounds, and methods of using them to treat tyrosine kinase-dependent diseases and conditions, such as angiogenesis, cancer, tumor growth, atherosclerosis, age related macular degeneration, diabetic retinopathy, inflammatory diseases, and the like in mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 20 OF 41 USPATFULL
ACCESSION NUMBER: 2002:266257 USPATFULL
TITLE: Compounds for targeting endothelial cells, compositions containing the same and methods for their use
INVENTOR(S): Von Wronski, Mathew A., Moorestown, NJ, UNITED STATES
Marinelli, Edmund R., Lawrenceville, NJ, UNITED STATES
Nunn, Adrian D., Lambertville, NJ, UNITED STATES
Pillai, Radhakrishna, Cranbury, NJ, UNITED STATES
Ramalingam, Kondareddiar, Dayton, NJ, UNITED STATES
Tweedle, Michael F., Princeton, NJ, UNITED STATES
Linder, Karen, Kingston, NJ, UNITED STATES
Nanjappan, Palaniappa, Dayton, NJ, UNITED STATES
Raju, Natarajan, Kendall Park, NJ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002147136	A1	20021010
APPLICATION INFO.:	US 2001-871974	A1	20010604 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-585364, filed on 2 Jun 2000, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NIXON & VANDERHYE P.C., 8th Floor, 1100 North Glebe Road, Arlington, VA, 22201-4714
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 5017
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides compounds for targeting endothelial cells, tumor cells or other cells that express the NP-1 receptor, compositions containing the same and methods for their use. Additionally, the present invention includes diagnostic, therapeutic and radiotherapeutic compositions useful for visualization, therapy or radiotherapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 21 OF 41 USPATFULL
ACCESSION NUMBER: 2002:165211 USPATFULL
TITLE: Use of Rad51 inhibitors for p53 gene therapy
INVENTOR(S): Zarling, David A., Menlo Park, CA, UNITED STATES
Reddy, Gurucharan, Fremont, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002086840	A1	20020704
APPLICATION INFO.:	US 2001-771355	A1	20010126 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-178561P	20000126 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FLEHR HOHBACH TEST, ALBRITTON & HERBERT LLP, Four Embarcarero Center, Suite 3400, San Francisco, CA, 94111-4187
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
LINE COUNT: 995
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to methods and compositions for

vivo. More specifically, a tumor cell is contacted, in vivo, with a Rad51 inhibitor, and a polynucleotide capable of expressing functional ***p53*** protein. In a further embodiment of the present invention the tumor cell is exposed in vivo to radiation or chemotherapeutic agents (e.g., BCNU, CCNU, and DMZ, GB, cisplatin and the like). The Rad51 inhibitor may be selected from the group consisting of peptides, small molecules and Rad51 ***antisense*** molecules. The Rad51 ***antisense*** molecule and the ***p53*** polynucleotide may be encoded on an expression vector under the control of one or more promoters, and the expression vector may then be incorporated into a viral genome, preferably an andeno or retro virus, which is then used to introduce the expression vector into the tumor cell.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 22 OF 41 USPATFULL
ACCESSION NUMBER: 2002:141539 USPATFULL
TITLE: Orally active salts with tyrosine kinase activity
INVENTOR(S): Fraley, Mark E., North Wales, PA, UNITED STATES
Karki, Shyam B., Lansdale, PA, UNITED STATES
Kim, Yuntae, Harleysville, PA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002072526	A1	20020613
APPLICATION INFO.:	US 2001-981979	A1	20011017 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-241043P	20001017 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MERCK AND CO INC, P O BOX 2000, RAHWAY, NJ, 070650907
NUMBER OF CLAIMS: 39
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 1971
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to orally active salts of compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which contain these compounds, and methods of using them to treat tyrosine kinase-dependent diseases and conditions, such as angio-genesis, cancer, tumor growth, atherosclerosis, age related macular degeneration, diabetic retinopathy, inflammatory diseases, and the like in mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 23 OF 41 USPATFULL
ACCESSION NUMBER: 2002:297601 USPATFULL
TITLE: Tyrosine kinase inhibitors
INVENTOR(S): Fraley, Mark E., North Wales, PA, United States
Hambaugh, Scott R., Norristown, PA, United States
Hungate, Randall W., Lansdale, PA, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6479512	B1	20021112
APPLICATION INFO.:	US 2000-690602		20001017 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-160362P	19991019 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Seaman, D. Margaret
LEGAL REPRESENTATIVE: Brown, Dianne, Daniel, Mark R.
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 2602
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which

kinase-dependent diseases and conditions, such as angiogenesis, cancer, tumor growth, atherosclerosis, age related macular degeneration, diabetic retinopathy, inflammatory diseases, and the like in mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 24 OF 41 USPATFULL
ACCESSION NUMBER: 2002:108831 USPATFULL
TITLE: ATM kinase modulation for screening and therapies
INVENTOR(S): Kastan, Michael, Cordova, TN, United States
Canman, Christine, Cordova, TN, United States
Kim, Seong-Tae, Cordova, TN, United States
Lim, Dae-Sik, Cordova, TN, United States
PATENT ASSIGNEE(S): St. Jude Children's Research Hospital, Memphis, TN, United States (U.S. corporation)
Johns-Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6387640	B1	20020514	
APPLICATION INFO.:	US 1999-248061		19990210	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Achutamurthy, Ponnathapu
ASSISTANT EXAMINER: Monshipouri, M.
LEGAL REPRESENTATIVE: Darby & Darby
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 2258
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to identification of the consensus sequence phosphorylated by ATM kinase. This, in turn, permitted identification of ATM kinase target proteins, and development of a convenient assay system for ATM kinase phosphorylation using fusion polypeptides as substrates. The assay system is adaptable to screening for ATM modulators, particularly inhibitors. In a specific embodiment, the substrate recognition sequence and mutagenized variants of this sequence were incorporated in a GST fusion protein and assayed for phosphorylation by ATM kinase. This assay system is useful in screening for ATM inhibitors. ATM function assays were validated using an ATM-kinase dead dominant-negative mutant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 25 OF 41 USPATFULL
ACCESSION NUMBER: 2002:69773 USPATFULL
TITLE: Non-invasive method for detecting target RNA
INVENTOR(S): Iversen, Patrick L., Corvallis, OR, United States
PATENT ASSIGNEE(S): AVI BioPharma, Inc., Corvallis, OR, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6365351	B1	20020402	
APPLICATION INFO.:	US 2000-493494		20000128	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-117846P	19990129 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Wang, Andrew
ASSISTANT EXAMINER: Zara, Jane
LEGAL REPRESENTATIVE: Judge, Linda R., Perkins Coie LLP
NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 3
NUMBER OF DRAWINGS: 11 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 1258
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method for targeting a particular mRNA sequence in vivo by oral administration of a ***morpholino*** antisense compound having uncharged phosphorus-containing backbone linkages. Also disclosed is a non-invasive method of detecting and quantitating the in vivo presence of RNA containing one or more selected

nuclease-resistant antisense oligomer which hybridizes by Watson-Crick base pairing to a region of the target RNA with a Tm substantially greater than 37.degree. C. The oligomer is able to complex intracellularly with target RNA, and is released from intracellular sites as a nuclease-resistant heteroduplex, which can then be measured in a body fluid sample, e.g., urine.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 26 OF 41 USPATFULL
ACCESSION NUMBER: 2002:34294 USPATFULL
TITLE: ATM kinase modulation for screening and therapies
INVENTOR(S): Kastan, Michael, Cordova, TN, United States
Canman, Christine, Cordova, TN, United States
Kim, Seong-Tae, Cordova, TN, United States
Lim, Dae-Sik, Cordova, TN, United States
PATENT ASSIGNEE(S): St. Jude Childre's Research Hospital, Memphis, TN, United States (U.S. corporation)
Johns-Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6348311	B1	20020219
APPLICATION INFO.:	US 1999-400653		19990921 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-248061, filed on 10 Feb 1999
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Prouty, Rebecca E.
ASSISTANT EXAMINER: Monshipouri, Maryam
LEGAL REPRESENTATIVE: Darby & Darby
NUMBER OF CLAIMS: 2
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 3229
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to identification of the consensus sequence phosphorylated by ATM kinase. This, in turn, permitted identification of ATM kinase target proteins, and development of a convenient assay system for ATM kinase phosphorylation using fusion polypeptides as substrates. The assay system is adaptable to screening for ATM modulators, particularly inhibitors. In a specific embodiment, the substrate recognition sequence and mutagenized variants of this sequence were incorporated in a GST fusion protein and assayed for phosphorylation by ATM kinase. This assay system is useful in screening for ATM inhibitors. ATM function assays were validated using an ATM-kinase dead dominant-negative mutant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 27 OF 41 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2002212307 MEDLINE
DOCUMENT NUMBER: 21945711 PubMed ID: 11948540
TITLE: Bioavailability and efficacy of antisense ***morpholino*** oligomers targeted to c-myc and cytochrome P-450 3A2 following oral administration in rats.
AUTHOR: Arora Vikram; Knapp Derek C; Reddy Muralimohan T; Weller Dwight D; Iversen Patrick L
CORPORATE SOURCE: AVI BioPharma, 4575 SW Research Way, Suite 200, Corvallis, Oregon 97333, USA.. varora@avibio.com
CONTRACT NUMBER: GM54871 (NIGMS)
SOURCE: JOURNAL OF PHARMACEUTICAL SCIENCES, (2002 Apr) 91 (4) 1009-18.
Journal code: 2985195R. ISSN: 0022-3549.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20020412
Last Updated on STN: 20020815
Entered Medline: 20020814
AB ***Antisense*** phosphorodiamidate ***Morpholino*** oligomers (PMO) are resistant to degradation by cellular hydrolases, DNases, RNases, and phosphodiesterases, but remain sensitive to prolonged exposure to low

stability, and efficacy of two distinct PMO sequences targeted to c-myc and cytochrome P-450 (CYP) 3A2. The c-myc ***antisense*** 20-mer, AVI-4126 (5'-ACGTTGAGGGGCATCGTCGC-3'), slowed the regenerative process in the rat liver after a 70% partial hepatectomy (PH). Rats were administered 3.0 mg/kg AVI-4126 in 0.1 mL saline via a bolus intravenous injection or in 0.5 mL sterile phosphate-buffered saline via gavage immediately following PH. The areas under the plasma concentration versus time curves revealed a fractional oral availability of 78.8% over a period of 10 min through 24 h. Immunoblot analysis of liver tissue from rats treated orally with AVI-4126 demonstrated a sequence-specific reduction in the target protein c-Myc, as well as secondary proliferation markers: proliferating cell nuclear antigen (PCNA), cyclin D1, and ***p53*** . The CYP3A2 ***antisense*** 22-mer AVI-4472 (5'-GAGCTGAAAGCAGGTCCATCCC-3') caused a sequence-dependent reduction of approximately five-fold in the rat liver CYP3A2 protein levels and erythromycin demethylation activity in 24 h following oral administration at a dose of 2 mg/kg. It is concluded that oral administration of PMOs can inhibit c-myc and CYP3A2 gene expression in rat liver by an ***antisense*** -based mechanism of action. These studies highlight the potential for development of PMOs as orally administered therapeutic agents.
 J Pharm Sci 91:1009-1018, 2002

L11 ANSWER 28 OF 41 CAPLUS
ACCESSION NUMBER: 2002:712518 CAPLUS
DOCUMENT NUMBER: 138:34532
TITLE: Responses of human cells to PAH-induced DNA damage
AUTHOR(S): Baird, William M.; Hooven, Louisa A.; Mahadevan, Brinda; Luch, Andreas; Seidel, Albrecht; Iversen, Patrick L.
CORPORATE SOURCE: Oregon State University, Corvallis, OR, 97331, USA
SOURCE: Polycyclic Aromatic Compounds (2002), 22(3-4), 771-780
CODEN: PARCEO; ISSN: 1040-6638
PUBLISHER: Taylor & Francis Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Benzo[a]pyrene (B[a]P) and dibenzo[a,l]pyrene (DB[a,l]P) induce cytochrome P 450 (CYP) CYP1A1 and CYP1B1, which metabolize these polycyclic arom. hydrocarbons (PAHs) into DNA-binding species. In order to detail roles of CYP1A1 and CYP1B1 in activation of DB[a,1]P to the diol epoxide, we here report the inhibition of CYP1A1 in human MCF-7 cells with phosphorodiamidate ***morpholino*** ***antisense*** oligomers (morpholinos). PAH-DNA adduct formation was also detd. after treatment with morpholinos and B[a]P or DB[a,l]P. ***P53*** is involved in DNA repair, cell cycle arrest, and apoptosis. Cells with normal ***p53*** protein arrest in the G1 phase of the cell cycle on exposure to DNA-damaging agents (presumably allowing the cell sufficient time to repair damaged DNA prior to replication). Previous studies in human MCF-7 cells indicate that cells with PAH-DNA adducts escape cell cycle arrest and accumulate in the S phase. In the present study the effect of PAH-DNA adducts on the cell cycle were obsd. in human diploid fibroblasts (HDF). We found that treatment of HDF with the diol epoxide of DB[a,l]P causes cell cycle arrest in G1. An increase in DNA adduct formation with increase in concn. of dibenzo[a,l]pyrene diol epoxide {(-)-anti-DB[a,l]PDE} was also obsd.
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 29 OF 41 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2002276603 MEDLINE
DOCUMENT NUMBER: 22011825 PubMed ID: 12015968
TITLE: A dominant-negative form of p63 is required for epidermal proliferation in zebrafish.
AUTHOR: Lee Hyunsook; Kimelman David
CORPORATE SOURCE: Department of Biochemistry, University of Washington, Box 357350, Seattle, WA 98195, USA.
SOURCE: Dev Cell, (2002 May) 2 (5) 607-16.
Journal code: 101120028. ISSN: 1534-5807.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF487944
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020518
Last Updated on STN: 20020618

AB Epidermal stem cells play a critical role in producing the multilayered vertebrate skin. Products of the p63 gene not only mark the epidermal stem cells, but also are absolutely required for the formation of mammalian epidermis. We find that early zebrafish embryos express a dominant-negative form of p63 (DeltaNp63), which accumulates in the nucleus just as epidermal growth begins. Using ***antisense*** ***morpholino*** oligonucleotides, we show that DeltaNp63 is needed for epidermal growth and limb development and is specifically required for the proliferation of epidermal cells by inhibiting ***p53*** activity. While the structure of fish epidermis is very different from that of higher vertebrates, our study shows that DeltaNp63 has essential and ancient role in the development of skin.

L11 ANSWER 30 OF 41 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2002628967 MEDLINE
DOCUMENT NUMBER: 22274651 PubMed ID: 12386920
TITLE: Inhibition of human chorionic gonadotropin beta-subunit modulates the mitogenic effect of c-myc in human prostate cancer cells.
AUTHOR: Devi Gayathri R; Oldenkamp Jennifer R; London Carla A; Iversen Patrick L
CORPORATE SOURCE: AVI BioPharma, Corvallis, Oregon 97333, USA.. grdevi@avibio.com
SOURCE: PROSTATE, (2002 Nov 1) 53 (3) 200-10. Journal code: 8101368. ISSN: 0270-4137.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20021019
Last Updated on STN: 20021213
Entered Medline: 20021121
AB BACKGROUND: Amplification of the proto-oncogene c-myc has been identified as one of the most common genetic alterations in prostate cancer, thus making it an attractive therapeutic target. However, certain prostate cancer cells are unresponsive to c-Myc inhibition. The purpose of this study was to test the hypothesis that effective growth inhibition in the refractory cancer cells can be achieved by blocking c-myc along with a growth factor using a novel phosphorodiamidate ***morpholino*** ***antisense*** oligomer-based approach. Human chorionic gonadotropin, a growth factor implicated in neoplasm, causes activation of c-myc through a G-protein-coupled pathway of signal transduction. METHODS: In this study, the effect of inhibition of beta-hCG and c-myc singly or in combination was evaluated in DU145 (RB -/-, ***p53*** -/-, androgen-independent) and LNCaP (Rb+/+, ***p53*** +/+, androgen-sensitive) human prostate cancer cell lines and in a DU145 subcutaneous xenograft murine model. RESULTS: ***Antisense*** phosphorodiamidate ***morpholino*** oligomers directed against beta-hCG and c-myc caused a specific decrease of the target protein levels. Unlike LNCaP cells, DU145 cell growth was refractory to c-Myc inhibition. Unresponsiveness to c-myc inhibition in DU145 cells was overcome by targeting both beta-hCG and c-myc genes, resulting in potentiation of the antiproliferative effect seen with inhibition of beta-hCG alone. CONCLUSIONS: The inhibition of beta-hCG sensitizes prostate cancer cells to the antiproliferative effects of c-Myc inhibition, including tumors that are refractory to c-Myc decrease alone. Copyright 2002 Wiley-Liss, Inc.

L11 ANSWER 31 OF 41 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:816897 CAPLUS
DOCUMENT NUMBER: 135:353717
TITLE: Splice-region antisense oligonucleotide composition and targeting the mRNA splicing
INVENTOR(S): Iversen, Patrick L.; Hudziak, Robert
PATENT ASSIGNEE(S): Avi Biopharma, Inc., USA
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001083740	A2	20011108	WO 2001-US14410	20010504
W: AU, CA, JP, KR				

PT, SE, TR
PRIORITY APPLN. INFO.: US 2000-202376P P 20000504
AB Antisense compns. targeted against an mRNA sequence for a selected protein, at a region having its 5' end from 1 to about 25 base pairs downstream of a normal splice acceptor junction in the preprocessed mRNA, are disclosed. The antisense compd. is Rnase-inactive, and is preferably a phosphorodiamidate-linked ***morpholino*** oligonucleotide. Such targeting is effective to inhibit natural mRNA splice processing, produce splice variant mRNAs, and inhibit normal expression of the protein.

L11 ANSWER 32 OF 41 USPATFULL
ACCESSION NUMBER: 2001:212454 USPATFULL
TITLE: Tyrosine kinase inhibitors
INVENTOR(S): Fraley, Mark E., North Wales, PA, United States
Hartman, George D., Lansdale, PA, United States
Hungate, Randall W., Newbury Park, CA, United States

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2001044451	A1	20011122	
	US 6420382	B2	20020716	
APPLICATION INFO.:	US 2001-788718	A1	20010220	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-185023P	20000225 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MERCK AND CO INC, P O BOX 2000, RAHWAY, NJ, 070650907
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
LINE COUNT: 2114
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which contain these compounds, and methods of using them to treat tyrosine kinase-dependent diseases and conditions, such as angiogenesis, cancer, tumor growth, atherosclerosis, age related macular degeneration, diabetic retinopathy, inflammatory diseases, and the like in mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 33 OF 41 USPATFULL
ACCESSION NUMBER: 2001:165573 USPATFULL
TITLE: Non-invasive method for detecting target RNA
INVENTOR(S): Iversen, Patrick L., Corvallis, OR, United States
PATENT ASSIGNEE(S): AVI BioPharma, Inc. (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2001024783	A1	20010927	
APPLICATION INFO.:	US 2000-736920	A1	20001213	(9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 2000-493494, filed on 28 Jan 2000, PENDING			

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-117846P	19990129 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IOTA PI LAW GROUP, 350 CAMBRIDGE AVENUE SUITE 250, P O BOX 60850, PALO ALTO, CA, 94306-0850
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 2004
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of detecting in a subject, the occurrence of a base-specific intracellular binding event involving a single-stranded target RNA, is disclosed. The method includes administering to the subject an oligomeric antisense compound having (i) from 8 to 40 bases, including a targeting base sequence that is complementary to a portion of the target RNA, (ii) a Tm, with respect to binding to a complementary RNA sequence, of greater than about 50.degree. C., and (iii) an ability to be actively taken up by mammalian cells, and (iv) conferring resistance of complementary RNA hybridized with the agent to RnaseH. Where the

substantially backbone. At a selected time after said administering the agent, a sample of a body fluid is obtained from the subject, and the presence in the sample of a nuclease-resistant heteroduplex composed of the antisense oligomer and the complementary portion of the target RNA is detected. The method is useful, for example, for detecting levels of gene expression, biochemical or physiological states that are characterized by expression of certain genes, genetic mutations, and the presence and identity of infective viral or bacterial agents. Also disclosed are arrays, kits and antibodies employed in carrying out the method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 34 OF 41 USPATFULL
ACCESSION NUMBER: 2001:139533 USPATFULL
TITLE: Antisense modulation of human MDM2 expression
INVENTOR(S): Miraglia, Loren J., Encinitas, CA, United States
Nero, Pamela, Oceanside, CA, United States
Graham, Mark J., San Clemente, CA, United States
Monia, Brett P., La Costa, CA, United States
Cowsert, Lex M., Carlsbad, CA, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001016575	A1	20010823
APPLICATION INFO.:	US 2001-752983	A1	20010102 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-280805, filed on 26 Mar 1999, GRANTED, Pat. No. US 6184212
Continuation-in-part of Ser. No. US 1998-48810, filed on 26 Mar 1998, GRANTED, Pat. No. US 6238921
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 E. Main Street, Marlton, NJ, 08053
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 3562
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compounds, compositions and methods are provided for inhibiting the expression of human mdm2. The compositions include antisense compounds targeted to nucleic acids encoding mdm2. Methods of using these oligonucleotides for inhibition of mdm2 expression and for treatment of diseases such as cancers associated with overexpression of mdm2 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 35 OF 41 USPATFULL
ACCESSION NUMBER: 2001:215034 USPATFULL
TITLE: Arteriovenous and venous graft treatments: methods and compositions
INVENTOR(S): Zalewski, Andrew, Elkins Park, PA, United States
Shi, Yi, Cheltenham, PA, United States
PATENT ASSIGNEE(S): Thomas Jefferson University, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6323184	B1	20011127
APPLICATION INFO.:	US 1995-424991		19950419 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 1994-US11853, filed on 17 Oct 1994 Continuation-in-part of Ser. No. US 1993-138637, filed on 15 Oct 1993, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Drinker Biddle & Reath LLP
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 58 Drawing Figure(s); 31 Drawing Page(s)
LINE COUNT: 2692
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method and compounds are provided for inhibiting the synthesis of extracellular matrix proteins. Compounds of the invention comprise oligonucleotides specific for nuclear proto-oncogenes. Preferably, oligonucleotides of the invention are selected from the group consisting

in the treatment of a variety of disorders, including sclerotic disorders and restenosis, associated with the inappropriate synthesis of extracellular matrix proteins, particularly collagen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 36 OF 41 USPATFULL
ACCESSION NUMBER: 2001:197035 USPATFULL
TITLE: Tyrosine kinase inhibitors
INVENTOR(S): Fraley, Mark E., North Wales, PA, United States
Hartman, George D., Lansdale, PA, United States
Hartman, Randall W., Newbury Park, CA, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6313138	B1	20011106	
	US 2001047007	A1	20011129	
APPLICATION INFO.:	US 2001-788720		20010220	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-185024P	20000225 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Dentz, Bernard
LEGAL REPRESENTATIVE: Garcia-Rivas, J. Antonio, Daniel, Mark R.
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
LINE COUNT: 2167
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which contain these compounds, and methods of using them to treat tyrosine kinase-dependent diseases and conditions, such as angiogenesis, cancer, tumor growth, atherosclerosis, age related macular degeneration, diabetic retinopathy, inflammatory diseases, and the like in mammals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 37 OF 41 USPATFULL
ACCESSION NUMBER: 2001:185309 USPATFULL
TITLE: Tyrosine kinase inhibitors
INVENTOR(S): Fraley, Mark E., North Wales, PA, United States
Arrington, Kenneth L., Elkins Park, PA, United States
Bilodeau, Mark T., Lansdale, PA, United States
Hartman, George D., Lansdale, PA, United States
Hoffman, William F., Lansdale, PA, United States
Kim, Yuntae, Harleysville, PA, United States
Hungate, Randall W., Newbury Park, CA, United States
PATENT ASSIGNEE(S): Merck & Co., Inc., Rahway, NJ, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6306874	B1	20011023	
APPLICATION INFO.:	US 2000-690598		20001017	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-160356P	19991019 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Shah, Mukund J.
ASSISTANT EXAMINER: Truong, Tamthom N.
LEGAL REPRESENTATIVE: Garcia-Rivas, J. Antonio, Daniel, Mark R.
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 3068
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to compounds which inhibit, regulate and/or modulate tyrosine kinase signal transduction, compositions which contain these compounds, and methods of using them to treat tyrosine kinase-dependent diseases and conditions, such as angiogenesis, cancer, tumor growth, atherosclerosis, age related macular degeneration,

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 38 OF 41 USPATFULL
ACCESSION NUMBER: 2001:78947 USPATFULL
TITLE: Antisense oligonucleotide modulation of human mdm2 expression
INVENTOR(S): Miraglia, Loren J., Encinitas, CA, United States
Nero, Pamela, Oceanside, CA, United States
Graham, Mark J., San Clemente, CA, United States
Monia, Brett P., La Costa, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals, Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6238921	B1	20010529	
APPLICATION INFO.:	US 1998-48810		19980326	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Schwartzman, Robert A.
ASSISTANT EXAMINER: Shibuya, Mark L.
LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
LINE COUNT: 1117
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compounds, compositions and methods are provided for inhibiting the expression of human mdm2. The compositions comprise antisense oligonucleotides targeted to nucleic acids encoding mdm2. Methods of using these oligonucleotides for inhibition of mdm2 expression and for treatment of diseases such as cancers associated with overexpression of mdm2 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 39 OF 41 USPATFULL
ACCESSION NUMBER: 2001:75543 USPATFULL
TITLE: Glucocorticoid receptor agonist and decreased PP5
INVENTOR(S): Honkanen, Richard E., Mobile, AL, United States
PATENT ASSIGNEE(S): South Alabama Medical Science Foundation, Mobile, AL, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6235891	B1	20010522	
APPLICATION INFO.:	US 1999-282736		19990331	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Yucel, Remy
ASSISTANT EXAMINER: Schmidt, M
LEGAL REPRESENTATIVE: Braman & Rogalskyj, LLP
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 5 Drawing Page(s)
LINE COUNT: 1893
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A composition comprises a glucocorticoid receptor agonist and a compound which decreases levels of active human serine/threonine protein phosphatase 5 protein in cells. The glucocorticoid receptor agonist is dexamethasone and the compound is an antisense oligonucleotide of about 8 to 50 nucleotides in length which is targeted to a nucleic acid encoding human serine/threonine protein phosphatase 5. The composition is useful in a method of enhancing glucocorticoid activity, and in a method of enhancing the inhibition of hyperproliferation of cells where the inhibition is by contacting the cells with a compound which decreases levels of active human serine/threonine protein phosphatase 5 protein in cells. The compound is thus useful to enhance glucocorticoid therapy and to enhance inhibition of hyperproliferation relating to PP5.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 40 OF 41 USPATFULL
ACCESSION NUMBER: 2001:18459 USPATFULL
TITLE: Antisense modulation of human mdm2 expression
INVENTOR(S): Miraglia, Loren J., Encinitas, CA, United States

Graham, Mark J., San Clemente, CA, United States
Monia, Brett P., La Costa, CA, United States
Cowsert, Lex M., Carlsbad, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6184212	B1	20010206
APPLICATION INFO.:	US 1999-280805		19990326 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1998-48810, filed on 26 Mar 1998
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: Epps, Janet
LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 2192
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Compounds, compositions and methods are provided for inhibiting the expression of human mdm2. The compositions include antisense compounds targeted to nucleic acids encoding mdm2. Methods of using these oligonucleotides for inhibition of mdm2 expression and for treatment of diseases such as cancers associated with overexpression of mdm2 are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 41 OF 41 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5
ACCESSION NUMBER: 2000:171770 CAPLUS
DOCUMENT NUMBER: 132:329861
TITLE: c-Myc antisense limits rat liver regeneration and indicates role for c-Myc in regulating cytochrome P-450 3A activity
AUTHOR(S): Arora, Vikram; Knapp, Derek C.; Smith, Barbara L.; Statdfield, Mary L.; Stein, David A.; Reddy, Muralimohan T.; Weller, Dwight D.; Iversen, Patrick L.
CORPORATE SOURCE: AVI BioPharma, Corvallis, OR, USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics (2000), 292(3), 921-928
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Expression of c-myc protein is assocd. with cell proliferation. The present study uses ***antisense*** oligomers to inhibit c-myc expression in the regenerating rat liver after 70% partial hepatectomy (PH). ***Antisense*** phosphorodiamidate ***morpholino*** oligomers (novel DNA analogs) were administered i.p. immediately after surgery to block expression of c-myc within the first 24 h after PH. A 20-mer PMO complimentary to the c-myc mRNA at the translation start site was an effective sequence (AVI-4126, 5'-ACGTTGAGGGGCATCGTCGC-3'). A single i.p. dose of 0.5 mg/kg AVI-4126 caused redn. of the regenerating liver c-myc protein in a sequence-specific and dose-dependent manner. Inhibition of c-myc expression resulted in redn. of proliferating cell nuclear antigen and arrested cells in the G0/G1 phase of the cell cycle. The ratio of G2:G0 cell populations in the regenerating liver 24 h after PH dropped from 29.1 in saline vehicle-treated rats to 18.0 in rats treated with 2.5 mg/kg AVI-4126. The expression of cell cycle checkpoint protein ***p53*** was inhibited with increasing doses of AVI-4126, but expression of p21waf-1 was unaffected. The activity of cytochrome P 450 3A2 (CYP3A2) was evaluated by immunoblot anal. and erythromycin N-demethylation. AVI-4126 did not alter CYP3A activity in nonhepatectamized animals but showed a dose-dependent decrease in PH rats. We conclude that AVI-4126, ***antisense*** oligomer to c-myc, can reduce cell proliferation in the regenerating rat liver. Furthermore, inhibition of c-myc may indirectly influence the expression of CYP3A.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d history

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     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     19:01:24 ON 21 JAN 2003
L1          4478 S P53 AND ANTISENSE
L2            78 S L1 AND SPLICE ACCEPTOR SITE
L3            78 DUP REM L2 (0 DUPLICATES REMOVED)
L4             2 S L3 AND MORPHOLINO
L5           171 S L1 AND MORPHOLINO
L6           157 DUP REM L5 (14 DUPLICATES REMOVED)
L7            37 S L6 AND PY<2001
L8          2189 S P53 (P) ANTISENSE
L9            66 S L8 AND MORPHOLINO
L10           54 S L9 NOT L7
L11           41 DUP REM L10 (13 DUPLICATES REMOVED)

=> s p53 (s) antisense
L12         1988 P53 (S) ANTISENSE

=>

=> d l12 1980-1988 ibib abs

L12  ANSWER 1980 OF 1988  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    1992:404269  BIOSIS
DOCUMENT NUMBER:     BR43:60144
TITLE:               TOXICITY OF HUMAN   ***P53***     ***ANTISENSE***
                     OLIGONUCLEOTIDE INFUSIONS IN RHESUS MACACA.
AUTHOR(S):           SPINOLO J; BAYEVER E; IVERSEN P; JOHANSSON S; CORNISH K;
                     PIRRUCELLO S; SMITH L; ARNESON M
CORPORATE SOURCE:    UNIV. NEBRASKA MED. CENTER, OMAHA, NEBR. 68198.
SOURCE:              83RD ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER
                     RESEARCH, SAN DIEGO, CALIFORNIA, USA, MAY 20-23, 1992. PROC
                     AM ASSOC CANCER RES ANNU MEET, (1992) 33 (0), 523.
                     CODEN: PAMREA.
DOCUMENT TYPE:       Conference
FILE SEGMENT:        BR; OLD
LANGUAGE:            English

L12  ANSWER 1981 OF 1988  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    1992:404268  BIOSIS
DOCUMENT NUMBER:     BR43:60143
TITLE:                 ***ANTISENSE***     ***P53***   OLIGODEOXYNUCLEOTIDES AS
                     POTENTIAL HUMAN ANTI-LEUKEMIC AGENTS.
AUTHOR(S):           BAYEVER E; HAINES K H; IVERSEN P L; SPINOLO J; KAY H D;
                     SMITH L
CORPORATE SOURCE:    UNIV. NEBRASKA MED. CENTER, OMAHA, NEBR. 68102.
SOURCE:              83RD ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER
                     RESEARCH, SAN DIEGO, CALIFORNIA, USA, MAY 20-23, 1992. PROC
                     AM ASSOC CANCER RES ANNU MEET, (1992) 33 (0), 523.
                     CODEN: PAMREA.
DOCUMENT TYPE:       Conference
FILE SEGMENT:        BR; OLD
LANGUAGE:            English

L12  ANSWER 1982 OF 1988  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    1992:404267  BIOSIS
DOCUMENT NUMBER:     BR43:60142
TITLE:               SYSTEMIC HUMAN   ***P53***     ***ANTISENSE***
                     OLIGONUCLEOTIDE IN RHESUS MONKEY.
AUTHOR(S):           IVERSEN P; CORNISH K; JOHANSSON S; FOY M; BERGOT J;
                     FREDIANI J; SMITH L; ARNESON M; BAYEVER E; SPINOLO J
CORPORATE SOURCE:    UNMC, OMAHA, NEBR. 68198.
SOURCE:              83RD ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER
                     RESEARCH, SAN DIEGO, CALIFORNIA, USA, MAY 20-23, 1992. PROC
                     AM ASSOC CANCER RES ANNU MEET, (1992) 33 (0), 522.
                     CODEN: PAMREA.
DOCUMENT TYPE:       Conference
FILE SEGMENT:        BR; OLD
LANGUAGE:            English

L12  ANSWER 1983 OF 1988  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    1992:336583  BIOSIS
DOCUMENT NUMBER:     BR43:26133
TITLE:                 ***ANTISENSE***     ***P53***   RNA REDUCES THE TUMOR
                     SUPPRESSOR FUNCTION IN HUMAN LUNG CANCER CELL LINES
                     CARRYING WILD TYPE OR MUTATED   ***P53***   GENE.
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CORPORATE SOURCE: DEP. THORACIC SURGERY, UNIV. TEX. M. D. ANDERSON CANCER CENT., HOUSTON, TEX. 77030.
SOURCE: KEYSTONE SYMPOSIUM ON GENE TRANSFER, REPLACEMENT AND AUGMENTATION, COPPER MOUNTAIN, COLORADO, USA, APRIL 3-9, 1992. J CELL BIOCHEM SUPPL, (1992) 0 (16 PART F), 50. CODEN: JCBSD7.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L12 ANSWER 1984 OF 1988 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1991:524536 BIOSIS
DOCUMENT NUMBER: BA92:135996
TITLE: RELEASE OF EARLY HUMAN HEMATOPOIETIC PROGENITORS FROM QUIESCENCE BY ANTISENSE TRANSFORMING GROWTH FACTOR BETA-1 OR RB OLIGONUCLEOTIDES.
AUTHOR(S): HATZFIELD J; LI M-L; BROWN E L; SOOKDEO H; LEVESQUE J-P; O'TOOLE T; GURNEY C; CLARK S C; HATZFELD A
CORPORATE SOURCE: LAB. C.N.R.S. BIOL. CELLULAIRE ET MOL. DES FACTEURS CROISSANCE, I.C.I.G., HOP. PAUL-BROUSSE, 94802 VILLEJUIF CEDEX, FRANCE.
SOURCE: J EXP MED, (1991) 174 (4), 925-930. CODEN: JEMEAV. ISSN: 0022-1007.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB We have used ***antisense*** oligonucleotides to study the roles of transforming growth factor .beta. (TGF-.beta.) and the two antioncogenes, retinoblastoma susceptibility (Rb) and ***p53*** , in the negative regulation of proliferation of early hematopoietic cells in culture. The ***antisense*** TGF-.beta. sequence significantly enhanced the frequency of colony formation by multi-lineage, early erythroid, and granulomonocytic progenitors, but did not affect colony formation by late progenitors. Single cell culture and limiting dilution analysis indicated that autocrine TGF-.beta. is produced by a subpopulation of early progenitors. ***Antisense*** Rb but not ***antisense*** ***p53*** yielded similar results in releasing multipotential progenitors (colony-forming unit-granulocyte/erythroid/macrophage/megakary ocyte) from quiescence. Rb ***antisense*** could partially reverse the inhibitory effect of exogenous TGF-.beta.. Anti-TGF-.beta. blocking antibodies, ***antisense*** TGF-.beta., or Rb oligonucleotides all had similar effects. No additive effects were observed when these reagents were combined, suggesting a common pathway of action. Our results are consistent with the model that autocrine production of TGF-.beta. negatively regulates the cycling status of early hematopoietic progenitors through interaction with the Rb gene product.

L12 ANSWER 1985 OF 1988 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1991:478184 BIOSIS
DOCUMENT NUMBER: BA92:111944
TITLE: COOPERATIVE EFFECT OF ***ANTISENSE*** -RB AND ***ANTISENSE*** - ***P53*** OLIGOMERS ON THE EXTENSION OF LIFE SPAN IN HUMAN DIPLOID FIBROBLASTS TIG-1.
AUTHOR(S): HARA E; TSURUI H; SHINOZAKI A; NAKADA S; ODA K
CORPORATE SOURCE: BIOLOGICAL SCI. TECHNOL., SCI. UNIV. TOKYO, CHIBA 278, JPN.
SOURCE: BIOCHEM BIOPHYS RES COMMUN, (1991) 179 (1), 528-534. CODEN: BBRCA9. ISSN: 0006-291X.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Normal human diploid fibroblasts, TIG-1, which have a replicative life span of about 62 population doublings (PD), tended to senesce after about 50 PD with a gradual decrease in sensitivity to serum. Treatment of TIG-1 cells with the ***antisense*** -Rb oligomer, which completely depleted the retinoblastoma susceptibility gene product (RB), extended life span by about 10 PD. Treatment with the ***antisense*** - ***p53*** oligomer alone had no effect; however, cotreatment with the ***antisense*** -Rb oligomer further potentiated the extension and the increased sensitivity to serum caused by the ***antisense*** -Rb oligomer alone, suggesting that ***p53*** and RB function in separate, yet complementary pathways in signal transduction to senescence. The c-fos expression, which is presumed to be regulated negatively by RB, was not stimulated in partially senescent TIG-1 cells by treatment with the ***antisense*** -Rb oligomer.

L12 ANSWER 1986 OF 1988 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1990:90746 BIOSIS
DOCUMENT NUMBER: BA89:50097

MESSENGER RNA MATURATION IN MURINE ERYTHROLEUKEMIA CELLS INDUCED TO DIFFERENTIATE.

AUTHOR(S): KHOCHBIN S; LAWRENCE J-J

CORPORATE SOURCE: LAB. DE BIOL. MOL. DU CYCLE CELLULAIRE, UNITE INSERM 309, DEP. DE RECHERCHE FONDAMENTALE, CEN-GRENOBLE, FRANCE.

SOURCE: EMBO (EUR MOL BIOL ORGAN) J, (1989) 8 (13), 4107-4114.
CODEN: EMJODG. ISSN: 0261-4189.

FILE SEGMENT: BA; OLD

LANGUAGE: English

AB A post-transcriptional control of gene expression was found to be responsible for a down-regulation of ***p53*** mRNA accompanying the induced differentiation of murine erythroleukemia (MEL) cells. Such a posttranscriptional control was governed by the induced synthesis of an RNA species (inRNA). In an attempt to find a potential candidate for such as function, we have localized the post-transcriptional regulation of ***p53*** mRNA in the nuclear compartment of the cells; then various fragments of the ***p53*** gene were used as probes for induced RNA(s) susceptible to interacting with ***p53*** pre-mRNA. This experimental approach allowed for the identification of a nuclear RNA molecule .apprx. 1.3 kb long, which was recognized specifically by a PstI-HindIII fragment located in the 5' part of the first intervening sequence of the ***p53*** gene. This RNA accumulated when cells were treated by the inducer concomitantly with high mol. wt ***p53*** mRNA precursors. However this RNA was not a maturation product of ***p53*** pre-mRNA as evidenced by its ***antisense*** orientation with respect to this RNA. Moreover it was markedly enriched in the poly(A)+ fraction. The complementary part of inRNA in the ***p53*** gene has been sequenced over .apprx. 1200 bp; no extensive homology was found in gene data banks but three restricted areas of the sequence were found homologous to a limited number of genes; they were themselves partially homologous to known repetitive sequences. Possible implication of such a sequence in the regulation of ***p53*** gene expression is discussed.

L12 ANSWER 1987 OF 1988 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1989:74802 BIOSIS

DOCUMENT NUMBER: BA87:39200

TITLE: FUNCTIONAL ROLE OF BK VIRUS TUMOR ANTIGENS IN TRANSFORMATION.

AUTHOR(S): NAKSHATRI H; PATER M M; PATER A

CORPORATE SOURCE: BASIC MED. SCI., FAC. MED., MEMORIAL UNIV. NEWFOUNDLAND, ST. JOHN'S, NEWFOUNDLAND, CAN. A1B 3V6.

SOURCE: J VIROL, (1988) 62 (12), 4613-4621.
CODEN: JOVIAM. ISSN: 0022-538X.

FILE SEGMENT: BA; OLD

LANGUAGE: English

AB We have examined the role of the human papovavirus BK virus (BKV) tumor (T) antigen(s) in the maintenance of transformation and have identified the domain of T antigen essential for transformation. BKV-transformed BHK 21 and NIH 3T3 cells expressing ***antisense*** T-antigen RNA lose their ability to grow in soft agar, indicating the need for the continued expression of T antigen for the maintenance of the transformed phenotype. Experiments using translation termination linker insertion and deletion mutagenesis of BKV T antigen demonstrate that amino acids 356 to 384 are essential for transformation. Although BKV T antigen shares 100, 95, and 82% amino acid homology with that of simian virus 40 (SV40) for the nuclear localization signal, ***p53*** -binding domain, and DNA-binding domain, respectively, the transformation domains of BKV and SV40 T antigens share only 54% homology. Also, BKV T antigen lacks a substantial portion of the ATPase domain of SV40, and our results indicate the dispensability of the remaining portion for transformation by this protein. We suggest that the differences in the amino acids in the identified transformation domains together with the differences in the ATPase domains may account for the differences in the transformation potentials of the two proteins.

L12 ANSWER 1988 OF 1988 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1988:374619 BIOSIS

DOCUMENT NUMBER: BA86:58529

TITLE: CONSTITUTIVE EXPRESSION OF C-FOS ANTISENSE RNA BLOCKS C-FOS GENE INDUCTION BY INTERFERON AND BY PHORBOL ESTER AND REDUCES C-MYC EXPRESSION IN F9 EMBRYONAL CARCINOMA CELLS.

AUTHOR(S): LEVI B-Z; OZATO K

CORPORATE SOURCE: LAB. DEV. MOL. IMMUNITY, NATL. INST. CHILD HEALTH HUM. DEV., NATL. INST. HEALTH, BETHESDA, MD. 20892, USA.

SOURCE: GENES DEV, (1988) 2 (5), 554-566.
CODEN: GEDEEP. ISSN: 0890-9369.

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LANGUAGE:          English
AB   To address the role of c-fos proto-oncogene we constructed a plasmid that
     allows constitutive expression of RNA complementary to c-fos mRNA, and
     stably introduced this plasmid into F9 embryonal carcinoma cells. Some F9
     clones expressing c-fos   ***antisense***   RNA had a reduced basal level
     of c-fos mRNA, and were unable to induce a c-fos mRNA as well as its
     protein when stimulated with phorbol ester or with interferon (IFN).
     Nevertheless, the ability to induce major histocompatibility class I genes
     following IFN treatment was not impaired in these clones. Clones
     expressing c-fos   ***antisense***   RNA grew as rapidly as control F9
     cells, and underwent differentiation after retinoic acid treatment.
     Unexpectedly, constitutive expression of c-myc mRNA was reduced on average
     by 10-fold in clones expressing c-fos   ***antisense***   RNA. However,
     expression of the   ***p53***   gene and heat shock gene hsp70 was not
     affected in these clones, indicating the existence of a specific
     regulatory linkage between c-fos and c-myc genes. Cycloheximide treatment
     led to induction of a large amount of c-fos mRNA in clones expressing
     c-fos   ***antisense***   RNA as well as in control F9 clones. The amount
     of c-fos   ***antisense***   RNA was also increased by cycloheximide
     treatment. We postulate that c-fos   ***antisense***   RNA blocks
     expression of the endogenous c-fos gene by accelerating the degradation of
     c-fos mRNA and that cycloheximide treatment interferes with this
     degradation.


=> e iversen ?/au
E1           1     IVERSBY SIDSEL/AU
E2           2     IVERSEN/AU
E3           0 --> IVERSEN ?/AU
E4          50     IVERSEN A/AU
E5           8     IVERSEN A A/AU
E6          18     IVERSEN A D/AU
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E9           1     IVERSEN A H/AU
E10          1     IVERSEN A J/AU
E11         20     IVERSEN A K/AU
E12         41     IVERSEN A K N/AU

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E1          10     IVERSEN P S/AU
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E3           0 --> IVERSEN P?/AU
E4           1     IVERSEN PAL MO/AU
E5           1     IVERSEN PALLE/AU
E6           1     IVERSEN PALLE BIRGER/AU
E7          31     IVERSEN PALLE E/AU
E8           3     IVERSEN PAT/AU
E9           1     IVERSEN PATICK L/AU
E10          1     IVERSEN PATRIC/AU
E11          4     IVERSEN PATRIC L/AU
E12         42     IVERSEN PATRICK/AU

=> s e8-e12
L13         51 ("IVERSEN PAT"/AU OR "IVERSEN PATICK L"/AU OR "IVERSEN PATRIC"/A
               U OR "IVERSEN PATRIC L"/AU OR "IVERSEN PATRICK"/AU)

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14          39 DUP REM L13 (12 DUPLICATES REMOVED)

=> s l14 and p53
L15          1 L14 AND P53

=> d l15 ibib

L15  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:        1994:208076  CAPLUS
DOCUMENT NUMBER:         120:208076
TITLE:                   Cardiovascular effects of a phosphorothioate
                         oligonucleotide with sequence antisense to   ***p53***
                         in the conscious rhesus monkey
AUTHOR(S):               Cornish, Kurtis G.;   ***Iversen, Patrick***  ; Smith,
                         Larry; Arneson, Mark; Bayever, Eliel
CORPORATE SOURCE:        Coll. Med., Univ. Nebraska, Omaha, NE, 68198-4575, USA
SOURCE:                  Pharmacology Communications (1993), 3(3), 239-47
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DOCUMENT TYPE: Journal
LANGUAGE: English

=> FIL STNGUIDE
COST IN U.S. DOLLARS — SINCE FILE ENTRY: 227.49 — TOTAL SESSION: 227.70

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) — SINCE FILE ENTRY: -9.77 — TOTAL SESSION: -9.77

FILE 'STNGUIDE' ENTERED AT 19:24:57 ON 21 JAN 2003

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jan 17, 2003 (20030117/UP).

=> d abs
YOU HAVE REQUESTED DATA FROM FILE 'CAPLUS' - CONTINUE? (Y)/N:y

L15 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2003 ACS
AB Antisense phosphorothioate oligonucleotides directed at ***p53*** mRNA selectively reduce renewal of human Acute Myelogenous Leukemic (AML) stem cells in culture. This would suggest that these cells could potentially be eliminated in-vivo. However, in-vitro data cannot easily be extrapolated to the intact animal. One concern relates to untoward side effects which cannot be examd. in-vitro. The objective of this study was to det. any cardiovascular alterations in the conscious primate following administration of a phosphorothioate oligonucleotide. The specific sequence, 5'-CCCTGCTCCCCCCTGGCTCC-3' (designated OL(1) ***p53***), is complementary to the mRNA encoding a 53 kDa protein called ***p53*** . Seven Rhesus monkeys (Macaca mulatta) were given 5 to 150 mg/kg OL(1) ***p53*** (five infusions less than 4 h and four infusions of greater than 6 days) while cardiovascular parameters were continuously monitored. Peak plasma oligonucleotide concns. were 0.37 to 0.77 .mu.M (.beta.-elimination phase) for short infusions, and 1.31 to 4.39 .mu.M for long infusions. The monkeys tolerated the treatment well and did not show any cardiovascular effects either during or after the infusion of OL(1) ***p53*** . The authors conclude that the slow administration of OL(1) ***p53*** to the conscious primate does not produce alterations in cardiovascular function.

=> e hudziak ?/au
'AU' IS NOT A VALID EXPAND FIELD CODE FOR FILE 'STNGUIDE'
The indicated field code is not available for EXPAND in this file. To see a list of valid EXPAND field codes, enter HELP SFIELDS at an arrow prompt (=>).

=> b medline caplus lifesci embase uspatfull biosis
COST IN U.S. DOLLARS — SINCE FILE ENTRY: 0.06 — TOTAL SESSION: 229.70

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) — SINCE FILE ENTRY: 0.00 — TOTAL SESSION: -10.42

FILE 'MEDLINE' ENTERED AT 19:27:06 ON 21 JAN 2003

FILE 'CAPLUS' ENTERED AT 19:27:06 ON 21 JAN 2003

FILE 'LIFESCI' ENTERED AT 19:27:06 ON 21 JAN 2003

FILE 'EMBASE' ENTERED AT 19:27:06 ON 21 JAN 2003

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FILE 'USPATFULL' ENTERED AT 19:27:06 ON 21 JAN 2003
CA INDEXING COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 19:27:06 ON 21 JAN 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

=> e hudziak ?/au
E1            1     HUDZI IVANKA LJ/AU
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E8            9     HUDZIAK J/AU
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E8            3     HUDZIEC PIOTR/AU
E9            5     HUDZIK A/AU
E10           1     HUDZIK ANDRZEJ/AU
E11           1     HUDZIK ANTONI/AU
E12           1     HUDZIK BARTOSZ/AU

=> s e1-e6
L16          90 ("HUDZIAK R"/AU OR "HUDZIAK R M"/AU OR "HUDZIAK R?"/AU OR "HUDZI
                AK ROBERT"/AU OR "HUDZIAK ROBERT M"/AU OR "HUDZIAK ROBERT MICHAE
                L"/AU)

=> s l16 and p53
L17           1 L16 AND P53

=> d l17 ibib abs

L17  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2001:816897  CAPLUS
DOCUMENT NUMBER:          135:353717
TITLE:                    Splice-region antisense oligonucleotide composition
                          and targeting the mRNA splicing
INVENTOR(S):              Iversen, Patrick L.;   ***Hudziak, Robert***
PATENT ASSIGNEE(S):       Avi Biopharma, Inc., USA
SOURCE:                   PCT Int. Appl., 53 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2001083740      A2   20011108       WO 2001-US14410  20010504
         W:  AU, CA, JP, KR
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, TR
PRIORITY APPLN. INFO.:                    US 2000-202376P  P  20000504
AB   Antisense compns. targeted against an mRNA sequence for a selected
     protein, at a region having its 5' end from 1 to about 25 base pairs
     downstream of a normal splice acceptor junction in the preprocessed mRNA,
     are disclosed.  The antisense compd. is Rnase-inactive, and is preferably
     a phosphorodiamidate-linked morpholino oligonucleotide.  Such targeting is
     effective to inhibit natural mRNA splice processing, produce splice
     variant mRNAs, and inhibit normal expression of the protein.


=> s p53 and exon()skipping
L1           25 P53 AND EXON(W) SKIPPING
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=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 13 DUP REM L1 (12 DUPLICATES REMOVED)

=> d l2 ibib abs tot

L2 ANSWER 1 OF 13 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002072617 MEDLINE
DOCUMENT NUMBER: 21657315 PubMed ID: 11799138
TITLE: Splicing mutations in TP53 in human squamous cell carcinoma lines influence immunohistochemical detection.
AUTHOR: Eicheler Wolfgang; Zips Daniel; Dorfler Annegret; Grenman Reidar; Baumann Michael
CORPORATE SOURCE: Department of Radiotherapy and Radiation Oncology, Turku University, Turku, Finland.. wolfgang.eicheler@mailbox.tu-dresden.de
SOURCE: JOURNAL OF HISTOCHEMISTRY AND CYTOCHEMISTRY, (2002 Feb) 50 (2) 197-204.
Journal code: 9815334. ISSN: 0022-1554.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200203
ENTRY DATE: Entered STN: 20020125
Last Updated on STN: 20020320
Entered Medline: 20020319

AB The mutational status of the tumor suppressor gene TP53 is often examined by immunohistochemistry. We compared the incidence of TP53 mutations in 12 permanent squamous cell carcinoma lines of the head and neck with the immunohistochemical staining obtained with two different antibodies. The mutational status of the TP53 gene was assessed by sequencing the complete coding frame of the TP53 mRNA. All 12 tumor cell lines had TP53 mutations. Six of them showed missense mutations and five had premature stop codons caused either by splicing mutations or nonsense mutations or by ***exon*** ***skipping*** . One tumor cell line was heterozygous, with a truncating splicing mutation and an additional missense mutation located on different alleles. In one case, an in-frame insertion of 23 extra codons was found. All missense mutations were positive in immunhistochemistry and Western blotting. The truncated ***p53*** was not immunohistochemically detected in three cases with the DO-7 antibody and in five cases with the G59-12 antibody, giving false-negative results in 25% or 40%, respectively, of all tumor cell lines examined. We conclude that splicing mutations are common in squamous cell carcinoma lines and that the incidence of ***p53*** inactiviation by erroneous splicing is higher than yet reported. Sequencing of only the exons of TP53 may miss intronic mutations leading to missplicing and may therefore systematically underestimate the TP53 mutation frequency.

L2 ANSWER 2 OF 13 USPATFULL
ACCESSION NUMBER: 2001:116764 USPATFULL
TITLE: Ataxia-telangiectasia gene and its genomic organization
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6265158	B1	20010724	
	WO 9636691		19961121	
APPLICATION INFO.:	US 1998-952014		19980202	(8)
	WO 1996-US7025		19960516	
			19980202	PCT 371 date
			19980202	PCT 102(e) date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996, now patented, Pat. No. US 5858661
Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Goldberg, Jeanine
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1,7

LINE COUNT: 3109
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia, its genomic organization, methods for the detection of the defective gene, the purified polypeptide encoded by the defective gene, and antibodies recognizing the defective protein.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 3 OF 13 USPATFULL

ACCESSION NUMBER:	2001:48208 USPATFULL			
TITLE:	Ataxia-telangiectasia gene			
INVENTOR(S):	Shiloh, Yosef, Tel Aviv, Israel Tagle, Danilo A., Gaithersburg, MD, United States Collins, Francis, Rockville, MD, United States			
PATENT ASSIGNEE(S):	The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government) Ramot University Authority for Applied Research and Industrial Dev., Israel (non-U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6211336	B1	20010403	
	WO 9636695		19961121	
APPLICATION INFO.:	US 1998-952127		19980226	(8)
	WO 1996-US7040		19960516	
			19980226	PCT 371 date
			19980226	PCT 102(e) date
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1995-508836, filed on 28 Jul 1995, now patented, Pat. No. US 5777093 Continuation-in-part of Ser. No. US 1995-493092, filed on 21 Jun 1995, now patented, Pat. No. US 5728807 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Cintins, Marianne M.			
ASSISTANT EXAMINER:	Delacroix-Muirheid, C.			
LEGAL REPRESENTATIVE:	Kohn & Associates			
NUMBER OF CLAIMS:	5			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	10 Drawing Figure(s); 4 Drawing Page(s)			
LINE COUNT:	2279			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB There is provided a purified amino acid sequence selected from the group of Sequence ID No.: 3 and analogs thereof and mutations of Sequence ID No.: 3 which cause ataxia-telangiectasia. Also provided is a purified amino acid sequence as set forth in Sequence ID No.: 3 and analogs thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 4 OF 13 USPATFULL

ACCESSION NUMBER:	2001:36598 USPATFULL			
TITLE:	Mutated forms of the ataxia-telangiectasia gene and method to screen for a partial A-T phenotype			
INVENTOR(S):	Shiloh, Yosef, Tel Aviv, Israel			
PATENT ASSIGNEE(S):	Ramot-University Authority for Applied Research and Industrial Development Ltd., Tel Aviv, Israel (non-U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6200749	B1	20010313	
APPLICATION INFO.:	US 1996-642274		19960503	(8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-629001, filed on 8 Apr 1996 Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288			
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Arthur, Lisa B.			
LEGAL REPRESENTATIVE:	Kohn & Associates			
NUMBER OF CLAIMS:	5			
EXEMPLARY CLAIM:	1,4			

LINE COUNT: 3090
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A purified and isolated gene, designated ATM, is described mutations of which cause ataxia-telangiectasia and its genomic organization.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 5 OF 13 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000497773 MEDLINE
DOCUMENT NUMBER: 20438181 PubMed ID: 10980610
TITLE: Detection of PTEN nonsense mutation and psiPTEN expression in central nervous system high-grade astrocytic tumors by a yeast-based stop codon assay.
AUTHOR: Zhang C L; Tada M; Kobayashi H; Nozaki M; Moriuchi T; Abe H
CORPORATE SOURCE: Section of Neurosurgery, Department of Neuropathophysiology, Hokkaido University Graduate School of Medicine, Sapporo, 060-8638 Japan.
SOURCE: ONCOGENE, (2000 Sep 7) 19 (38) 4346-53.
Journal code: 8711562. ISSN: 0950-9232.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200010
ENTRY DATE: Entered STN: 20001027
Last Updated on STN: 20001027
Entered Medline: 20001019
AB We have developed a new yeast-based assay for the detection of PTEN nonsense mutation, and applied it to a total of 42 astrocytic tumors. The assay utilizes homologous recombination of PCR-amplified PTEN cDNA samples to a yeast vector which expresses an in-frame PTEN::ADE2 chimera protein. An allele of nonsense mutation in the sample PTEN mRNA gives a truncated chimera protein in a yeast cell, resulting in the formation of a red colony. The assay and subsequent sequence analysis demonstrated nonsense mutations as red colonies of more than 10% in one of 10 anaplastic astrocytomas and six of 18 glioblastomas, but none in six pilocytic astrocytomas or in eight astrocytomas. Sequence analysis of white colonies showed one missense mutation in a glioblastoma. Interestingly, four of seven nonsense mutations were frame-shifts due to ***exon*** ***skipping*** . In addition, pink colonies were found in one of six pilocytic astrocytomas, three of eight astrocytomas, two of 10 anaplastic astrocytomas, and 10 of 18 glioblastomas. Sequence analysis of the pink colonies revealed a sequence similar to those reported as psiPTEN/PTH2. By testing mRNA and genomic DNA, it was found to be a processed pseudogene which was transcribed. The psiPTEN expression was complementary to PTEN mutation, for 14 of 18 glioblastomas showed either PTEN mutation or psiPTEN expression and only one case showed both PTEN mutation and psiPTEN expression ($P<0.046$), suggesting a pathological role of psiPTEN expression as an alternative to PTEN mutation in glioblastomas.

L2 ANSWER 6 OF 13 MEDLINE
ACCESSION NUMBER: 2000307560 MEDLINE
DOCUMENT NUMBER: 20307560 PubMed ID: 10848880
TITLE: FHIT and TSG101 in thyroid tumours: aberrant transcripts reflect rare abnormal RNA processing events of uncertain pathogenetic or clinical significance.
AUTHOR: McIver B; Grebe S K; Wang L; Hay I D; Yokomizo A; Liu W; Goellner J R; Grant C S; Smith D I; Eberhardt N L
CORPORATE SOURCE: Department of Medicine, Mayo Clinic/Foundation, New Zealand.
SOURCE: CLINICAL ENDOCRINOLOGY, (2000 Jun) 52 (6) 749-57.
Journal code: 0346653. ISSN: 0300-0664.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000811
Last Updated on STN: 20000811
Entered Medline: 20000803
AB OBJECTIVE: The chromosomal regions containing the two putative tumour suppressors, fragile histidine triad gene (FHIT) and tumour suppressor gene 101 (TSG101), are deleted frequently in thyroid tumours. We therefore analysed FHIT and TSG101 transcripts in a group of advanced thyroid tumours to establish their role in thyroid tumorigenesis. DESIGN: Retrospective analysis of FHIT and TSG101 mRNA transcripts and genomic DNA

level not to be mutated in this cohort of tumours, served as a control. PATIENTS: We analysed nine follicular thyroid carcinomas (FTC), six papillary thyroid carcinomas and six follicular adenomas (FA) and histologically normal thyroid tissue from four of the FA patients. MEASUREMENTS: Single stage and nested reverse transcription polymerase chain reaction (RT-PCR) products of FHIT, TSG101, and TP53 were analysed by agarose or polyacrylamide gel electrophoresis and sequenced. Genomic DNA was also analysed by polymerase chain reaction and sequencing (FHIT) or by Southern blotting (TSG101). Clinical data were correlated with the results of the mutation analysis. RESULTS: Truncated FHIT transcripts were observed frequently alongside full length transcripts with nested RT-PCR, most often in FTC, while single stage RT-PCR revealed only normal length transcripts in all tumours. Similar results were obtained for TP53, while abnormal TSG101 transcripts were detectable by single stage RT-PCR. Sequence analysis of the truncated FHIT and TSG101 transcripts revealed mainly ***exon*** ***skipping*** and alternate RNA processing events. Only a single point mutation (of TSG101) was found. Southern blotting for the TSG101 gene, and PCR amplification and sequencing of the FHIT gene showed no evidence of genomic abnormalities in either case, and there was no evidence of splice site mutations in the FHIT gene, suggesting that the truncated transcripts result from altered RNA processing. There was no relationship between tumour stage, grade or survival and the presence of FHIT or TSG101 abnormalities. CONCLUSIONS: Truncated FHIT and TSG101 transcripts in thyroid tumours reflect alternate mRNA splicing events, rather than genomic deletions. Such abnormal RNA processing seems to be common and widespread in thyroid neoplasms, as similar results were obtained by analysis of transcripts of TP53, which we had previously shown not to be mutated in these specimens. Although a pathogenetic role for these aberrant transcripts remains possible, no correlation was found with stage, histological grade or outcome in this small group of advanced thyroid malignancies. Relaxation of mRNA splice control appears to be a feature of follicular cell-derived thyroid neoplasms.

L2 ANSWER 7 OF 13 USPATFULL
ACCESSION NUMBER: 1999:16738 USPATFULL
TITLE: Computational analysis of nucleic acid information defines binding sites
INVENTOR(S): Schneider, Thomas D., Frederick, MD, United States
Rogan, Peter K., Lebanon, PA, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5867402		19990202	
APPLICATION INFO.:	US 1995-494115		19950623	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Ramirez, Ellis B.
ASSISTANT EXAMINER: Kemper, M.
LEGAL REPRESENTATIVE: Morgan & Finnegan, L.L.P.
NUMBER OF CLAIMS: 57
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 30 Drawing Figure(s); 22 Drawing Page(s)
LINE COUNT: 1944
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In accordance with the present invention, binding sites are defined based upon the individual information content of a particular site of interest. Substitutions within the binding site sequences can be analyzed to determine whether the substitution will cause a deleterious mutation or a benign polymorphism. In addition, new binding sites can be identified using individual information content. Further a computer system is described for determining and displaying individual information content of a binding site sequence.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 8 OF 13 USPATFULL
ACCESSION NUMBER: 1999:4329 USPATFULL
TITLE: Ataxia-telangiectasia gene and its genomic organization
INVENTOR(S): Shiloh, Yosef, Tel Aviv, Israel
PATENT ASSIGNEE(S): RAMOT-University Authority for Applied Research and Industrial Development, Tel Aviv, Israel (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5858661		19990112
APPLICATION INFO.:	US 1996-629001		19960408 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-441822, filed on 16 May 1995, now patented, Pat. No. US 5756288
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Arthur, Lisa B.
LEGAL REPRESENTATIVE: Kohn & Associates
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1,7
NUMBER OF DRAWINGS: 6 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 3461
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A purified and isolated gene, designated ATM, mutations of which cause ataxia-telangiectasia and its genomic organization is disclosed. Methods and a kit for the detection of carriers of mutations of the ATM gene are provided by analysis of nucleic acids isolated from patients including in situ hybridization, Northern blotting and reverse transcriptase--polymerase chain reaction, Southern blotting, single strand conformational polymorphism, restriction endonuclease fingerprinting (REF), PCR amplification and DNA-chip analysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 9 OF 13 USPATFULL
ACCESSION NUMBER: 97:56495 USPATFULL
TITLE: Methods for diagnosing cancer, precancerous state, or susceptibility to other forms of diseases by detecting an acceleration of ***exon*** ***skipping*** in IRF-1 mRNA
INVENTOR(S): Taniguchi, Tadatsugu, Ibaraki, Japan
Harada, Hisashi, Suita, Japan
PATENT ASSIGNEE(S): Boehringer Ingelheim International GmbH, Germany, Federal Republic of (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5643729		19970701
APPLICATION INFO.:	US 1995-393997		19950224 (8)

	NUMBER	DATE
PRIORITY INFORMATION:	EP 1994-102839	19940224

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Zitomer, Stephanie W.
ASSISTANT EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 963
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention concerns a novel molecular marker useful for diagnosing hematopoietic disorders, including cancers and precancerous conditions. The invention is based on the unexpected discovery that inactivation of the IRF-1 tumor suppressor gene can occur via an altered splicing pattern of the IRF-1 primary transcript. This altered splicing pattern leads to mRNAs lacking exon 2 or exons 2 and 3. The relative amounts of full-length RNA and shortened RNA molecules are significantly different in samples obtained from patients suffering from certain cancers and precancerous conditions as compared to healthy donors.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 10 OF 13 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97479031 MEDLINE
DOCUMENT NUMBER: 97479031 PubMed ID: 9337692
TITLE: An intronic deletion in TP53 gene causes exon 6 skipping in breast cancer.
AUTHOR: Voglino G; Castello S; Silengo L; Stefanuto G; Friard O; Ferrara G; Fessia L
CORPORATE SOURCE: Department of Clinical Pathology, Ospedale Sant' Anna, Turin, Italy.

Journal code: 9005373. ISSN: 0959-8049.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971103

AB Six hundred and thirty primary breast cancer were screened for abnormalities in exons 5, 6, 7 and 8 of the TP53 tumour suppressor gene. Analysis of the structure of the TP53 gene exons was performed with the polymerase chain reaction-single strand conformation polymorphism (PCR-SSCP) method and with direct sequencing of amplified DNA. In a breast tumour case from a postmenopausal patient, we found a deletion of 36 bp in intron 5 and no immunohistochemical staining for ***p53*** . We amplified and sequenced the cDNA region between exons 4 and 7 and showed that the deletion causes the skipping of exon 6. The resulting mRNA sequence had a frameshift that yields an inactive protein with a truncated C terminus. These results show the first example of intronic deletion causing ***exon*** ***skipping*** at the TP53 gene level.

L2 ANSWER 11 OF 13 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 96382695 MEDLINE
DOCUMENT NUMBER: 96382695 PubMed ID: 8790559
TITLE: Alterations of the RB tumour suppressor gene in hepatocellular carcinoma and hepatoblastoma cell lines in association with abnormal ***p53*** expression.
AUTHOR: Farshid M; Hsia C C; Tabor E
CORPORATE SOURCE: National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA.
SOURCE: JOURNAL OF VIRAL HEPATITIS, (1994) 1 (1) 45-53.
Journal code: 9435672. ISSN: 1352-0504.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961025
Last Updated on STN: 19970203
Entered Medline: 19961017

AB Alterations in the expression of the RB tumour suppressor gene were found by Western immunoblot in three of seven hepatocellular carcinoma and hepatoblastoma cell lines. Abnormalities were detected by single-strand conformation polymorphism (SSCP) within exons 17-21 in RB cDNA from two of these three cell lines and within exons 20-21 in the third cell line. In these three cell lines with abnormal RB expression, abnormal expression of the ***p53*** tumour suppressor gene was also found, apparently the product of a mutant gene. Thus, mutations within the RB gene (or splice-site mutations with ***exon*** - ***skipping***) and apparent mutations of the ***p53*** gene together may have contributed to the development of three of these tumours or to the establishment of these cell lines.

L2 ANSWER 12 OF 13 USPATFULL
ACCESSION NUMBER: 92:65884 USPATFULL
TITLE: Methods and compositions for the detection of sequences in selected DNA molecules
INVENTOR(S): LeMaistre, Anne, Humble, TX, United States
Lee, Ming-Shen, Houston, TX, United States
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5137806		19920811
APPLICATION INFO.:	US 1989-448118		19891211 (7)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Wax, Robert A.
ASSISTANT EXAMINER: Escallon, Miguel
LEGAL REPRESENTATIVE: Arnold, White & Durkee
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 1498

AB The present disclosure relates to novel procedures and primers for use in conenction with PCR or in vitro DNA sequence amplification to detect sequence variants, such as sequence modifications or mutations. The invention will have particular applicability in the detection of point or other relatively short mutations where the expected location or configuration of the mutation is known. Primers of the invention incorporate a 3' terminal nucleotide or nucleotides complementary to the sequence variance, and thereby serve to successfuly prime chain elongation only on DNA templates which include the particular variant. Exemplary mutations suitable for detection through practice of the invention include those involved in beta-thalassemia, sickle cell anemia, hemoglobin C disease, diabetes, acute intermittent porphyria, lung, breast, and colon cancers and others.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 13 OF 13 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 93201632 MEDLINE
DOCUMENT NUMBER: 93201632 PubMed ID: 1295697
TITLE: Alternatively-spliced ***p53*** mRNA in the FAA-HTC1 rat hepatoma cell line without the splice site mutations.
AUTHOR: Fukuda I; Ogawa K
CORPORATE SOURCE: Department of Pathology, Asahikawa Medical College, Japan.
SOURCE: CELL STRUCTURE AND FUNCTION, (1992 Dec) 17 (6) 427-32. Journal code: 7608465. ISSN: 0386-7196.
PUB. COUNTRY: Japan
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-S47136; GENBANK-S47137; GENBANK-S47164; GENBANK-S47165; GENBANK-S47166; GENBANK-S47167; GENBANK-S47168; GENBANK-S51472; GENBANK-S57234; GENBANK-S72771
ENTRY MONTH: 199304
ENTRY DATE: Entered STN: 19930507
Last Updated on STN: 19970203
Entered Medline: 19930420
AB A novel mutation of the ***p53*** gene has been found in a rat hepatoma cell line, FAA-HTC1. This cell line carried two kinds of abnormal ***p53*** transcripts; one lacked the exon 8 sequence, and the other had a single base substitution G to T which resulted in a new stop codon in exon 8. In the genomic DNA, this base substitution in exon 8 was present, indicating that both transcripts were transcribed from the mutated gene. No mutation was detected in its two flanking introns. In this cell line, the exon-deleted transcript seems to be generated by ***exon*** ***skipping*** due to an unknown mechanism other than splice site mutations.

=> kwic 12
KWIC IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> d kwic 12

L2 ANSWER 12 OF 13 USPATFULL
SUMM . . . cystic fibrosis and Huntington's chorea. Furthermore, cancer oncogenes such as N-ras, K-ras, H-ras, Neu, or tumor suppressor genes such as ***p53*** are known to have such mutations which contribute to the cancer development. In the case of sickle cell disease, the. . .
DETD . . . disease (Grandchamp, et al.: A point mutation G to A in exon 12 of the porphobilinogen deaminase gene results in ***exon*** ***skipping*** and is responsible for acute intermittent prophyria. Nucleic acids research 17:6637-49, 1989). As in example I a primer construct can. . .

=> s p53 and antisense and (exon or intron)
L3 1379 P53 AND ANTISENSE AND (EXON OR INTRON)

=> s p53 same antisense same (exon or intron)
MISSING OPERATOR 'SAME (EXON'
The search profile that was entered contains terms or

```
=> s p53 (p) antisense (p)(exon or intron)
L4          57 P53 (P) ANTISENSE (P)(EXON OR INTRON)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5          32 DUP REM L4 (25 DUPLICATES REMOVED)

=> d l5 ibib abs tot

L5   ANSWER 1 OF 32  USPATFULL
ACCESSION NUMBER:        2002:251087  USPATFULL
TITLE:                   METHOD OF DETECTION OF NEOPLASTIC CELLS
INVENTOR(S):             SIDRANSKY, DAVID, BALTIMORE, MD, UNITED STATES
                         BAYLIN, STEPHEN B., BALTIMORE, MD, UNITED STATES
PATENT ASSIGNEE(S):      JOHN HOPKINS UNIVERSITY SCHOOL OF MEDICINE (U.S.
                         corporation)

                              NUMBER        KIND    DATE
                         --------------- ------ --------
PATENT INFORMATION:      US 2002137030      A1   20020926
APPLICATION INFO.:       US 1999-225904     A1   19990105  (9)
RELATED APPLN. INFO.:    Division of Ser. No. US 1995-497535, filed on 30 Jun
                         1995, PATENTED
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    LISA A. HAILE, PH.D., GRAY CARY WARE & FREIDENRICH LLP,
                         4365 EXECUTIVE DRIVE, SUITE 1600, SAN DIEGO, CA,
                         92121-2189
NUMBER OF CLAIMS:        11
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      11 Drawing Page(s)
LINE COUNT:              2249
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB       Methylation of p16 DNA and a resultant decrease in p16 gene expression
         is associated with transcriptional block and is associated with a
         variety of neoplasms. A method for detecting a neoplasm in a subject by
         detecting methylation of 5'CpG islands in p16 DNA, or detecting p16 mRNA
         or polypeptide levels in a sample is also provided. 51

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5   ANSWER 2 OF 32  USPATFULL
ACCESSION NUMBER:        2002:303980  USPATFULL
TITLE:                   Modification of mutated P53 gene in tumors by
                         retroviral delivery of ribozyme A
INVENTOR(S):             Roth, Jack A., Houston, TX, United States
                         Cai, De Wei, Cheltenham, PA, United States
                         Mukhopadhyay, Tapas, Houston, TX, United States
PATENT ASSIGNEE(S):      Board of Regents, The University of Texas System,
                         Austin, TX, United States (U.S. corporation)

                              NUMBER        KIND    DATE
                         --------------- ------ --------
PATENT INFORMATION:      US 6482803         B1   20021119
APPLICATION INFO.:       US 1995-523030          19950901  (8)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            GRANTED
PRIMARY EXAMINER:        LeGuyader, John L.
LEGAL REPRESENTATIVE:    Fulbright & Jaworski
NUMBER OF CLAIMS:        25
EXEMPLARY CLAIM:         1,4
NUMBER OF DRAWINGS:      12 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT:              2784
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB       The present invention discloses expression constructs and methods for
         employing them that result in the modulation of abnormal oncogene and
         tumor suppressor genes in a novel approach to cancer prevention and
         therapy. In one embodiment, an expression construct expresses a ribozyme
         that inactivates mutant p53 and also expresses the functional p53.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5   ANSWER 3 OF 32  USPATFULL
ACCESSION NUMBER:        2002:152389  USPATFULL
TITLE:                   Box-dependent Myc-interacting protein (Bin1)
```

INVENTOR(S): Prendergast, George C., Bala Cynwyd, PA, United States
Sakamuro, Daitoku, West Lafayette, IN, United States
PATENT ASSIGNEE(S): The Wistar Institute of Anatomy and Biology, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6410238	B1	20020625	
	WO 9855151		19981210	
APPLICATION INFO.:	US 1999-445247		19991203	(9)
	WO 1998-US11647		19980604	
			19991203	PCT 371 date

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-870126, filed on 6 Jun 1997, now patented, Pat. No. US 6048702
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Howson and Howson
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 2809
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides Bin1 genomic sequences and proteins encoded thereby. Also provided are compositions and methods utilizing these sequences and proteins in the diagnosis and treatment of cancers and hyperplastic disease states. Further provided are oligonucleotides derived from sequences encoding Bin1, as well as compositions and methods utilizing same for diagnostic and therapeutic purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 4 OF 32 USPATFULL
ACCESSION NUMBER: 2002:69973 USPATFULL
TITLE: p53 antisense agent and method
INVENTOR(S): Iversen, Patrick L., Corvallis, OR, United States
PATENT ASSIGNEE(S): AVI BioPharma, Inc., Corvallis, OR, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6365577	B1	20020402	
APPLICATION INFO.:	US 1999-426804		19991022	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-105695P	19981026 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Wang, Andrew
ASSISTANT EXAMINER: Zara, Jane
LEGAL REPRESENTATIVE: Gorthey, LeeAnn
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 1006
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense oligonucleotides useful for treating a disease state characterized by p53 induction, such as proliferative cell disorders, e.g. cancer, or a hypoxic state induced by an ischemic attack, such as stroke, are described. The antisense agents are preferably of the class known as "steric blocker" type oligonucleotides, including morpholino oligonucleotides, peptide nucleic acids, 2'-O-allyl or 2'-O-alkyl modified oligonucleotides, or N3'.fwdarw.P5' phosphoramidate oligonucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 5 OF 32 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002473200 MEDLINE
DOCUMENT NUMBER: 22220365 PubMed ID: 12235210
TITLE: DeltaNp73, a dominant-negative inhibitor of wild-type p53 and TAp73, is up-regulated in human tumors.
AUTHOR: Zaika Alex I; Slade Neda; Erster Susan H; Sansome Christine; Joseph Troy W; Pearl Michael; Chalas Eva; Moll Ute M

Brook, NY 11794, USA.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (2002 Sep 16) 196 (6) 765-80.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 20020918
Last Updated on STN: 20021011
Entered Medline: 20021010

AB p73 has significant homology to ***p53*** . However, tumor-associated up-regulation of p73 and genetic data from human tumors and p73-deficient mice exclude a classical Knudson-type tumor suppressor role. We report that the human TP73 gene generates an NH(2) terminally truncated isoform. DeltaNp73 derives from an alternative promoter in ***intron*** 3 and lacks the transactivation domain of full-length TAp73. DeltaNp73 is frequently overexpressed in a variety of human cancers, but not in normal tissues. DeltaNp73 acts as a potent transdominant inhibitor of wild-type ***p53*** and transactivation-competent TAp73. DeltaNp73 efficiently counteracts transactivation function, apoptosis, and growth suppression mediated by wild-type ***p53*** and TAp73, and confers drug resistance to wild-type ***p53*** harboring tumor cells. Conversely, down-regulation of endogenous DeltaNp73 levels by ***antisense*** methods alleviates its suppressive action and enhances ***p53*** - and TAp73-mediated apoptosis. DeltaNp73 is complexed with wild-type ***p53*** , as demonstrated by coimmunoprecipitation from cultured cells and primary tumors. Thus, DeltaNp73 mediates a novel inactivation mechanism of ***p53*** and TAp73 via a dominant-negative family network. Deregulated expression of DeltaNp73 can bestow oncogenic activity upon the TP73 gene by functionally inactivating the suppressor action of ***p53*** and TAp73. This trait might be selected for in human cancers.

L5 ANSWER 6 OF 32 USPATFULL
ACCESSION NUMBER: 2001:182581 USPATFULL
TITLE: Methods for delivering compounds into a cell
INVENTOR(S): Unger, Evan C., Tucson, AZ, United States
McCreery, Thomas, Tucson, AZ, United States
PATENT ASSIGNEE(S): ImaRX Pharmaceutical Corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001031740	A1	20011018
APPLICATION INFO.:	US 2000-742938	A1	20001221 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1997-841169, filed on 29 Apr 1997, PENDING Continuation-in-part of Ser. No. US 1997-785661, filed on 17 Jan 1997, ABANDONED Continuation-in-part of Ser. No. US 1996-640554, filed on 1 May 1996, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Woodcock Washburn Kurtz, MacKiewicz & Norris LLP, One Liberty Place - 46th Floor, Philadelphia, PA, 19103
NUMBER OF CLAIMS: 104
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 2971
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed, inter alia, to a method for delivering a compound into a cell comprising administering to the cell the compound to be delivered, an organic halide, and/or a carrier. Ultrasound may also be applied, if desired.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 7 OF 32 USPATFULL
ACCESSION NUMBER: 2001:231267 USPATFULL
TITLE: Promoter smooth muscle cell expression
INVENTOR(S): Parmacek, Michael S., Bryn Mawr, PA, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE

APPLICATION INFO.: US 1999-431349 19991101 (9)
RELATED APPLN. INFO.: Division of Ser. No. US 1996-726807, filed on 7 Oct 1996

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-4868P	19951005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Fulbright & Jaworski, LLP
NUMBER OF CLAIMS: 75
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 3926
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 8 OF 32 USPATFULL
ACCESSION NUMBER: 2001:215223 USPATFULL
TITLE: Transgenic mouse models for human bladder cancer
INVENTOR(S): Wu, Xue-Ru, Woodside, NY, United States
Sun, Tung-Tien, Scarsdale, NY, United States
PATENT ASSIGNEE(S): New York University, New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6323390	B1	20011127

APPLICATION INFO.: US 1998-83541 19980522 (9)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-969315, filed on 13 Nov 1997 Continuation-in-part of Ser. No. US 1997-907800, filed on 8 Aug 1997, now patented, Pat. No. US 6001646 Continuation-in-part of Ser. No. US 1995-464961, filed on 5 Jun 1995, now patented, Pat. No. US 5824543
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Martin, Jill D.
LEGAL REPRESENTATIVE: Browdy & Neimark
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 16 Drawing Page(s)
LINE COUNT: 1344
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A transgenic mouse, containing an oncogene or a tumor suppressor gene operably linked to a urothelium-specific promoter in its germ cells and somatic cells serves as an animal model system for human bladder cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 9 OF 32 USPATFULL
ACCESSION NUMBER: 2001:168105 USPATFULL
TITLE: Method for promoting angiogenesis with a nucleic acid construct comprising an SM22.alpha.0 promoter
INVENTOR(S): Parmacek, Michael S., Chicago, IL, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6297221	B1	20011002

APPLICATION INFO.: US 1999-225670 19990105 (9)
RELATED APPLN. INFO.: Division of Ser. No. US 1996-726807, filed on 7 Oct 1996, now patented, Pat. No. US 6090618
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED

LEGAL REPRESENTATIVE: Fulbright & Jaworski, LLP
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 3718
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 10 OF 32 USPATFULL
ACCESSION NUMBER: 2001:158043 USPATFULL
TITLE: Promoter for smooth muscle cell expression
INVENTOR(S): Parmacek, Michael S., Chicago, IL, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6291211	B1	20010918
APPLICATION INFO.:	US 1999-431414		19991101 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1996-726807, filed on 7 Oct 1996, now patented, Pat. No. US 6090618

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-4868P	19951005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Fulbright & Jaworkski, LLP
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 27 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 3788
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 11 OF 32 USPATFULL
ACCESSION NUMBER: 2001:152683 USPATFULL
TITLE: Early detection of ovarian carcinoma using p16 gene products
INVENTOR(S): O'Brien, Timothy J., Little Rock, AR, United States
Shiqemasa, Kazushi, Hiroshima, Japan
PATENT ASSIGNEE(S): Board of Trustees of the University of Arkansas, Little Rock, AR, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6287775	B1	20010911
APPLICATION INFO.:	US 1999-346200		19990701 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1997-819358, filed on 17 Mar 1997 Continuation of Ser. No. US 1996-621180, filed on 21 Mar 1996

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-41554P	19960321 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McKelvey, Terry

LEGAL REPRESENTATIVE: Adler, Benjamin Aaron
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 441
AB Increased expression of the p16 gene occurs early in the development of ovarian carcinomas. This invention detects change ovarian epithelium by measuring increases in p16 gene expression by a quantitative method that compares the levels of p16 mRNA and a control mRNA (.beta.-tubulin) in a subject to be tested against the levels of these substrates in normal subjects. A biological sample such as peritoneal fluid containing mRNA derived from a subject's ovarian epithelium is taken from the subject to be tested. The mRNA is isolated from the sample, and complementary cDNA is prepared from the isolated mRNA. Using primers to p16 target sequences and to .beta.-tubulin control sequences, the cDNA is amplified. The resultant amplification products are quantitated as to p16 and .beta.-tubulin gene sequences. The level p16 gene expression is assessed relative to expression levels in normal subjects. An increased level of p16 gene expression in a subject determined by this method is an indication of change in the subject's ovarian epithelium indicative of presence of a carcinoma.

L5 ANSWER 12 OF 32 USPATFULL
ACCESSION NUMBER: 2001:147951 USPATFULL
TITLE: Method for modulating smooth muscle cell proliferation
INVENTOR(S): Parmacek, Michael S., Chicago, IL, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6284743	B1	20010904
APPLICATION INFO.:	US 2000-546550		20000410 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-258367, filed on 26 Feb 1999, now patented, Pat. No. US 6114311 Division of Ser. No. US 1996-726807, filed on 7 Oct 1996, now patented, Pat. No. US 6090618

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-4868P	19951005 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Fulbright & Jaworski, LLP
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1,2
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 3738
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 13 OF 32 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001112715 MEDLINE
DOCUMENT NUMBER: 20576421 PubMed ID: 11013255
TITLE: Cloning and characterization of the human activity-dependent neuroprotective protein.
AUTHOR: Zamostiano R; Pinhasov A; Gelber E; Steingart R A; Seroussi E; Giladi E; Bassan M; Wollman Y; Eyre H J; Mulley J C; Brenneman D E; Gozes I
CORPORATE SOURCE: Department of Clinical Biochemistry, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv 69978, Israel.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2001 Jan 5) 276 (1) 708-14.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States

LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF250860
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

AB We have recently cloned the mouse activity-dependent neuroprotective protein (ADNP). Here, we disclose the cloning of human ADNP (hADNP) from a fetal brain cDNA library. Comparative sequence analysis of these two ADNP orthologs indicated 90% identity at the mRNA level. Several single nucleotide polymorphic sites were noticed. The deduced protein structure contained nine zinc fingers, a proline-rich region, a nuclear bipartite localization signal, and a homeobox domain profile, suggesting a transcription factor function. Further comparative analysis identified an ADNP paralog (33% identity and 46% similarity), indicating that these genes belong to a novel protein family with a nine-zinc finger motif followed by a homeobox domain. The hADNP gene structure spans approximately 40 kilobases and includes five exons and four introns with alternative splicing of an untranslated second ***exon*** . The hADNP gene was mapped to chromosome 20q12-13.2, a region associated with aggressive tumor growth, frequently amplified in many neoplasias, including breast, bladder, ovarian, pancreatic, and colon cancers. hADNP mRNA is abundantly expressed in distinct normal tissues, and high expression levels were encountered in malignant cells. Down-regulation of ADNP by ***antisense*** oligodeoxynucleotides up-regulated the tumor suppressor ***p53*** and reduced the viability of intestinal cancer cells by 90%. Thus, ADNP is implicated in maintaining cell survival, perhaps through modulation of ***p53*** .

L5 ANSWER 14 OF 32 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 3
ACCESSION NUMBER: 2000:67424 CAPLUS
DOCUMENT NUMBER: 132:127702
TITLE: Inhibiting the growth of p53-deficient tumor cells by administering the p53 gene
INVENTOR(S): Roth, Jack A.; Mukhopadhyay, Tapas; Tainsky, Michael A.
PATENT ASSIGNEE(S): Board of Regents, the University of Texas System, USA
SOURCE: U.S., 37 pp., Cont.-in-part of U.S. Ser. No. 665,538, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6017524	A	20000125	US 1992-960513	19921013
CA 2108144	AA	19920907	CA 1992-2108144	19920306
US 6436700	B1	20020820	US 1992-987235	19921207
US 6410010	B1	20020625	US 1993-145826	19931029
US 6143290	A	20001107	US 1994-224232	19940407
US 5747469	A	19980505	US 1994-233002	19940425
US 6069134	A	20000530	US 1997-953290	19971017
US 6511847	B1	20030128	US 2000-668532	20000921
US 2003012770	A1	20030116	US 2002-170240	20020611

PRIORITY APPLN. INFO.: US 1991-665538 B2 19910306
US 1992-960513 A2 19921013
US 1993-145826 A3 19931029
US 1994-233002 A3 19940425

AB Disclosed are methods and compns. for the selective manipulation of gene expression through the prepn. of retroviral expression vectors for expressing ***antisense*** sequences, such as K-ras oncogene ***antisense*** sequences, or sequences encoding a desired product, such as wild type ***p53*** sequences. Preferred retroviral vectors of the present invention incorporate the .beta.-actin promoter in a reverse orientation with respect to retroviral transcription. Preferred ***antisense*** RNA constructs of the present invention employ the use of ***antisense*** ***intron*** DNA corresponding to distinct ***intron*** regions of the gene whose expression is targeted for down-regulation. In an exemplary embodiment, a human lung cancer cell line (NCI-H460a) with a homozygous spontaneous K-ras mutation was transfected with a recombinant plasmid that synthesizes a genomic segment of K-ras in ***antisense*** orientation. Translation of the mutated K-ras mRNA was specifically inhibited, whereas expression of H-ras and

cells when expression of the mutated ras p21 protein was down-regulated by ***antisense*** RNA and cells remained viable. The growth of H460a tumors in nu/nu mice was substantially reduced by expressed K-ras ***antisense*** RNA.
REFERENCE COUNT: 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 15 OF 32 USPATFULL
ACCESSION NUMBER: 2000:117692 USPATFULL
TITLE: Method for modulating smooth muscle cell proliferation
INVENTOR(S): Parmacek, Michael S., Chicago, IL, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6114311		20000905
APPLICATION INFO.:	US 1999-258367		19990226 (9)

RELATED APPLN. INFO.: Division of Ser. No. US 1996-726807, filed on 7 Oct 1996
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Arnold White & Durkee
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 4109
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 16 OF 32 USPATFULL
ACCESSION NUMBER: 2000:91773 USPATFULL
TITLE: DNA constructs and viral vectors comprising a smooth muscle promoter
INVENTOR(S): Parmacek, Michael S., Chicago, IL, United States
Solway, Julian, Glencoe, IL, United States
PATENT ASSIGNEE(S): Arch Development Corporation, Chicago, IL, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6090618		20000718
APPLICATION INFO.:	US 1996-726807		19961007 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: McKelvey, Terry
LEGAL REPRESENTATIVE: Arnold White & Durkee
NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 21 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 4310
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed is a smooth muscle cell specific promoter, the SM22.alpha. gene promoter as well as the murine cDNA and genomic SM22.alpha. nucleic acid sequences. Also disclosed are methods of preventing restenosis following balloon angioplasty and methods of treating asthma based on inhibition of smooth muscle cell proliferation by expressing cell cycle control genes, or contraction inhibiting peptides in smooth muscle cells, under the control of the SM22.alpha. promoter.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 17 OF 32 USPATFULL
ACCESSION NUMBER: 2000:43942 USPATFULL
TITLE: Murine and human box-dependent myc-interacting protein (Bin1) and uses therefor

Sakamuro, Daitoku, Philadelphia, PA, United States
PATENT ASSIGNEE(S): The Wistar Institute of Anatomy and Biology, Philadelphia, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6048702		20000411
APPLICATION INFO.:	US 1997-870126		19970606 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1996-652972, filed on 24 May 1996, now patented, Pat. No. US 5723581 which is a continuation-in-part of Ser. No. US 1995-435454, filed on 5 May 1995, now patented, Pat. No. US 5605830
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Guzo, David
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Howson and Howson
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 3611
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides antibodies raised against a Box-dependent myc-interacting polypeptide termed Bin1 or fragments thereof are provided. Also provided are compositions and methods utilizing these antibodies in the diagnosis and treatment of cancers and hyperplastic disease states. Further provided are oligonucleotides derived from sequences encoding Bin1, as well as compositions and methods utilizing same for diagnostic and therapeutic purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 18 OF 32 USPATFULL
ACCESSION NUMBER: 2000:21739 USPATFULL
TITLE: Transgenic animals overexpressing MDM2
INVENTOR(S): Wasylyk, Bohdan, Illkirsch, France
Tocque, Bruno, Courbevoie, France
Alkhalaf, Moussa, Renne, France
PATENT ASSIGNEE(S): Rhone-Poulenc Rorer SA, Antony Cedex, France (non-U.S. corporation)
Institut National de la Sante et de la Recherche Medicale, Paris, France (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6028245		20000222
APPLICATION INFO.:	US 1998-104497		19980625 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-51739P	19970703 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Campell, Bruce R.
ASSISTANT EXAMINER: Baker, Anne-Marie
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 32 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1268
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to transgenic, non-human animals overexpressing a MDM2 gene. These animals model MDM2 over-expression associated with human tumors, display a major phenotype characterized by the severe skin disorder ichthyosis, and are useful for identifying compounds for the treatment of human disease. Therefore, the invention also relates to methods of using the animals for identifying compounds effective for the treatment of diseases of the skin and respiratory tract, and to the compounds themselves.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 19 OF 32 USPATFULL
ACCESSION NUMBER: 2000:4947 USPATFULL
TITLE: MDM2-specific antisense oligonucleotides
INVENTOR(S): Chen, Jiandong, Metairie, LA, United States
Agrawal, Sudhir, Shrewsbury, MA, United States

PATENT ASSIGNEE(S): Hybridon, Inc., Cambridge, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6013786		20000111
APPLICATION INFO.:	US 1998-73567		19980506 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-916384, filed on 22 Aug 1997
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degen, Nancy
ASSISTANT EXAMINER: Wang, Andrew
LEGAL REPRESENTATIVE: McDonnell Boehnen Hulbert & Berghoff
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 21 Drawing Page(s)
LINE COUNT: 1809
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides methods to activate tumor suppressors. The invention further provides antisense oligonucleotides complementary to a portion of the MDM2-encoding RNA and methods for using such antisense oligonucleotides as analytical and diagnostic tools, as potentiators of transgenic animal studies and for gene therapy approaches, and as potential therapeutic agents. The invention also provides methods to augment and synergistically activate a tumor suppressor in conjunction with the use of a DNA-damage inducing agent.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 20 OF 32 USPATFULL
ACCESSION NUMBER: 1999:1434 USPATFULL
TITLE: Method of detection of neoplastic cells
INVENTOR(S): Sidransky, David, Baltimore, MD, United States
Baylin, Stephen B., Baltimore, MD, United States
PATENT ASSIGNEE(S): The Johns Hopkins University School of Medicine, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5856094		19990105
APPLICATION INFO.:	US 1995-497535		19950630 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1995-439962, filed on 12 May 1995, now patented, Pat. No. US 5767258
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Houtteman, Scott W.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 2257
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methylation of p16 DNA and a resultant decrease in p16 gene expression is associated with transcriptional block and is associated with a variety of neoplasms. A method for detecting a neoplasm in a subject by detecting methylation of 5'CpG islands in p16 DNA, or detecting p16 mRNA or polypeptide levels in a sample is also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 21 OF 32 USPATFULL
ACCESSION NUMBER: 1998:48564 USPATFULL
TITLE: P53AS protein and antibody therefor
INVENTOR(S): Kulesz-Martin, Molly F., Buffalo, NY, United States
PATENT ASSIGNEE(S): Health Research, Inc., Buffalo, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5747650		19980505
APPLICATION INFO.:	US 1996-644456		19960510 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-100496, filed on 2 Aug 1993
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted

ASSISTANT EXAMINER: Bansal, Geetha P.
LEGAL REPRESENTATIVE: Dunn, Michael L.
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 1580
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB In accordance with the present invention, we have discovered and purified a protein designated herein as p53as, which protein is present in normal cells of a mammal and is essentially identical to known normal growth controlling protein p53 of the same mammal, at least until the final 50 amino acids of the carboxy terminal end of the protein. The invention further includes an antibody specific for protein p53as, which antibody is designated herein as Ab p53as. The antibody may be either a monoclonal or polyclonal antibody and may be specific for p53as of any particular mammal such as mice and humans.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 22 OF 32 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1999053919 MEDLINE
DOCUMENT NUMBER: 99053919 PubMed ID: 9840184
TITLE: The presence of wild-type TP53 is necessary for the radioprotective effect of the Bowman-Birk proteinase inhibitor in normal fibroblasts.
AUTHOR: Dittmann K H; Gueven N; Mayer C; Ohneseit P; Zell R; Begg A C; Rodemann H P
CORPORATE SOURCE: Department of Radiotherapy, Eberhard-Karls-University, Tubingen, Germany.
SOURCE: RADIATION RESEARCH, (1998 Dec) 150 (6) 648-55.
Journal code: 0401245. ISSN: 0033-7587.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981214
AB In the present study we have demonstrated that the Bowman-Birk proteinase inhibitor (BBI) protected normal fibroblasts from a radiation-induced reduction in cell survival, whereas in transformed fibroblasts no radioprotective effect was observed. It was shown that BBI reduced the radiation-induced protein stabilization and DNA-binding activity of TP53 (formerly known as ***p53***) in normal fibroblasts. In transformed fibroblasts, BBI failed to induce these effects. The analysis of the TP53 gene in transformed fibroblasts revealed a mutation in ***exon*** 5. As a consequence of this mutation, the expression of the TP53 downstream gene CDKN1A (p21/WAF1/Cip1) is blocked. Based on experiments using TP53 ***antisense*** oligonucleotides, the radioprotective effect of BBI could be correlated with the function of wild-type TP53. Thus BBI can be considered as a selective radioprotective agent for normal human fibroblasts.

L5 ANSWER 23 OF 32 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 5
ACCESSION NUMBER: 1997:216970 CAPLUS
DOCUMENT NUMBER: 126:288623
TITLE: A sensitive and high-resolution method for detection of mutations in the p53 gene using multiple fluorescence-based symmetric PCR-SSCP analysis
AUTHOR(S): Katsuragi, Kiyonori; Chiba, Wataru; Ikeda, Sadao; Ueta, Chie; Kinoshita, Moritoshi
CORPORATE SOURCE: Diagnostics Division, Otsuka Pharmaceutical Co., Ltd., Tokushima, 771-01, Japan
SOURCE: Biomedical Research (1997), 18(1), 57-64
CODEN: BRESD5; ISSN: 0388-6107
PUBLISHER: Biomedical Research Foundation
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mutations of the ***p53*** gene are an important feature of neoplastic progression in humans. The presence of such mutations has been detected in exons 5 through 8, which contain 86% of all mutations reported for the ***p53*** gene. The authors have developed a simple and rapid method, multiple fluorescence-based sym. polymerase chain reaction in a single tube and single-strand conformation polymorphism anal. in one lane (MF-SPCR-SSCP) for detection of mutations in exons 5, 6, 7 and 8 of the

a single tube using mixed four-color fluorescence-labeled sense and ***antisense*** primers. This technique enabled labeling of each ***exon*** of the product with a unique fluorescent. The MF-SPCR products were heat-denatured and applied to 7% polyacrylamide gel contg. 5% glycerol set on an automated DNA sequencer with a gel temp.-controlling system. The authors analyzed 18 specimens of lung cancer tissue with mutations in exons 5 through 7 using MF-SPCR-SSCP method. These mutations were detected even with use of only one PCR and one set of conditions for electrophoresis.

L5 ANSWER 24 OF 32 USPATFULL
ACCESSION NUMBER: 96:120741 USPATFULL
TITLE: Methods and compositions for detecting base pair mismatches
INVENTOR(S): Winkler, Matthew, Austin, TX, United States
Goldrick, Marianna M., Pflugerville, TX, United States
PATENT ASSIGNEE(S): Ambion, Inc., Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5589329		19961231
APPLICATION INFO.:	US 1993-155937		19931115 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Jones, W. Gary
ASSISTANT EXAMINER: Marschel, Ardin H.
LEGAL REPRESENTATIVE: Arnold White & Durkee
NUMBER OF CLAIMS: 44
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Figure(s); 11 Drawing Page(s)
LINE COUNT: 1735
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention discloses improved compositions and methods for detecting mutations, including single base changes, in nucleic acid sequences using RNase protection assays. The improvements include concomitant, dramatic reductions in the salt and RNase enzyme concentrations in the RNase digestion reaction mixture which result in greater sensitivity in detecting genetic mutations. Another embodiment of the present invention is kits to be used for the detection of single base mismatches in nucleic acid samples.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 25 OF 32 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 6
ACCESSION NUMBER: 95354258 EMBASE
DOCUMENT NUMBER: 1995354258
TITLE: Role of p53 in MCF-10F cell immortalization and chemically-induced neoplastic transformation.
AUTHOR: Barnabas N.; Moraes R.; Calaf G.; Estrada S.; Russo J.
CORPORATE SOURCE: Breast Cancer Research Laboratory, Fox Chase Cancer Center, 7701 Burholme Avenue,Philadelphia, PA 19111, United States
SOURCE: International Journal of Oncology, (1995) 7/6 (1289-1296). ISSN: 1019-6439 CODEN: IJONES
COUNTRY: Greece
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The present study was undertaken to determine the role of the tumor suppressor gene ***p53*** in the transformation of the human breast epithelial cell (HBEC) line MCF-10F treated with chemical carcinogens in vitro. MCF-10F is a spontaneously immortalized diploid HBEC line, derived from a mortal cell strain designated MCF-10M. MCF-10F cells became neoplastically transformed by in vitro treatment with the chemical carcinogens 7, 12-dimethylbenz(a)anthracene (DMBA) and benzo(a)pyrene (BP). DMBA and BP-treated cells gave rise to clones D3, D3-1, BP1 and BP1-E, respectively, all of which expressed colony formation in agar-methocel and high chemoinvasion index. BP1-E cells, derived from BP1, were tumorigenic in severe combined immunodeficient (SCID) mice. We designed this work utilizing this model in which isolated clones of cells express different stages of progression to neoplastic transformation for determining whether any specific phenotype was associated with alteration in the ***p53*** tumor supressor gene. For this purpose, Southern blot, Northern blot, single-strand conformation polymorphism (SSCP) and DNA sequencing were used to detect mutations in the highly conserved exons

the cells tested by Southern and Northern blot, SSCP analysis showed a conformational shift in ***exon*** 7 in the MCF-10F cell line, and in clones BP1, BP1-E, D3, and D3-1, derived from DMBA and BP treated cells, respectively. This shift was absent in MCF-10M cells, the mortal cells from which the MCF-10F immortal cells were derived, and in the placental DNA used as control. Sequence analysis using asymmetric PCR-amplified products of ***exon*** 7 and an ***antisense*** primer revealed an insertional mutation of thymine at codon 254 in MCF-10F cells and in transformed cells, but not in MCF-10M. These data indicate that the emergence of the immortalized phenotype was associated with a mutation of ***p53*** . DMBA- or BP-treatment did not induce additional changes in the ***p53*** gene. The fact that the precursor of the immortalized MCF-10F did not present changes in ***p53*** , may indicate that the alteration of this tumor suppressor gene could be associated with the process of cell immortalization; this, in turn, might facilitate the neoplastic transformation of the cells by chemical carcinogens.

L5 ANSWER 26 OF 32 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 95186358 MEDLINE
DOCUMENT NUMBER: 95186358 PubMed ID: 7880719
TITLE: Antisense oligonucleotides directed against p53 have antiproliferative effects unrelated to effects on p53 expression.
AUTHOR: Barton C M; Lemoine N R
CORPORATE SOURCE: Imperial Cancer Research Fund Oncology Unit, Hammersmith Hospital, London, UK.
SOURCE: BRITISH JOURNAL OF CANCER, (1995 Mar) 71 (3) 429-37.
Journal code: 0370635. ISSN: 0007-0920.
PUB. COUNTRY: SCOTLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950425
Last Updated on STN: 19980206
Entered Medline: 19950412
AB ***Antisense*** oligonucleotides targeting ***p53*** have been hailed as a potentially new technique for treating patients with cancer, and there have been encouraging reports of good patient tolerance in vivo and of antiproliferative effects in vitro. However, evidence is lacking that these oligonucleotides are acting via an ***antisense*** interaction to modulate ***p53*** expression. We examined a phosphorothioate ***antisense*** oligonucleotide, directed against ***exon*** 10 of the TP53 gene, and a chimaeric phosphorothioate-phosphodiester oligonucleotide directed against the ***p53*** translation initiation codon. Both failed to specifically suppress ***p53*** protein production in a cell-free assay system or to have any effect on mutant ***p53*** expression by human pancreatic cancer cell lines. Antiproliferative effects were apparent, especially with the phosphorothioate ***antisense*** oligonucleotide, but this was independent of the ***p53*** status of the cells (mutant, wild-type or absent) and also occurred with the control (sense and randomised) oligonucleotides. The most dramatic antiproliferative effects were seen with the 'control' phosphorothioate oligonucleotides. These findings suggest that the antiproliferative effects of some ***antisense*** oligonucleotides may be unrelated to expression of the gene they have been designed to target.

L5 ANSWER 27 OF 32 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 92340559 MEDLINE
DOCUMENT NUMBER: 92340559 PubMed ID: 1378845
TITLE: Characterization of an endogenous RNA transcript with homology to the antisense strand of the human c-myc gene.
AUTHOR: Celano P; Berchtold C M; Kizer D L; Weeraratna A; Nelkin B D; Baylin S B; Casero R A Jr
CORPORATE SOURCE: Johns Hopkins Oncology Center Laboratories, Baltimore, Maryland 21231.
CONTRACT NUMBER: CA 51068 (NCI)
CA47492 (NCI)
CA51085 (NCI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1992 Jul 25) 267 (21) 15092-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

OTHER SOURCE: GENBANK-M63383; GENBANK-M86615; GENBANK-M86616; GENBANK-M86617; GENBANK-M86618; GENBANK-M86619; GENBANK-M86620; GENBANK-M91159; GENBANK-M91212; GENBANK-X62322
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920911
Last Updated on STN: 19970203
Entered Medline: 19920826

AB In addition to being regulated by a complex array of cis- and trans-acting factors, c-myc protooncogene expression may be modulated by ***antisense*** RNA transcripts. Our previous studies have determined that depletion of intracellular polyamines by alpha-difluoromethylornithine results in a marked decrease in the transcription of the human c-myc gene. Because of reports that ***antisense*** transcription occurs in the 5' and 3' regions of this gene, we used a genomic clone of the human c-myc gene to ascertain whether polyamine depletion might induce an ***antisense*** RNA transcript. These studies demonstrate that polyamine depletion of the human colon cancer cell line COLO 320 results in induction of an endogenous RNA transcript with high homology to the ***antisense*** strand of the second intervening sequence (PvuII-RsaI) of the c-myc gene. Furthermore, during such depletion, steady state levels of this transcript vary inversely to the sense direction c-myc RNA. RNase protection studies suggest that the ***antisense*** transcript may arise from a different gene locus than the c-myc gene. To further identify the origins of this RNA, a cDNA library was generated from size-selected RNA and screened with c-myc sequences. A 438-base pair cDNA was isolated with approximately 85% homology, to a 285-base region in the second ***intron*** of the c-myc gene. Computer homology analysis further reveals that a 120-base region within this cDNA also has approximately 85% homology to the ***antisense*** strands of a number of genes, including the growth-related genes, N-myc, ***p53*** , and thymidine kinase. These studies provide the initial characterization of an endogenous ***antisense*** RNA transcript which could influence cell growth by modulating the expression of c-myc and other genes.

L5 ANSWER 28 OF 32 MEDLINE DUPLICATE 9
ACCESSION NUMBER: 93104490 MEDLINE
DOCUMENT NUMBER: 93104490 PubMed ID: 1361370
TITLE: Transcription factors, translocations, and leukemia.
AUTHOR: Nichols J; Nimer S D
CORPORATE SOURCE: Department of Medicine, UCLA School of Medicine 90024-1678.
CONTRACT NUMBER: DK43025 (NIDDK)
SOURCE: BLOOD, (1992 Dec 15) 80 (12) 2953-63. Ref: 118
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199301
ENTRY DATE: Entered STN: 19930212
Last Updated on STN: 19950206
Entered Medline: 19930125

AB The frequent occurrence of TF gene involvement in translocations associated with leukemia is remarkable, although not yet explained. The wide variety of TFs involved in these translocations and the different stages of cellular maturation argue against a unifying mechanism. Recombinases, active during B-cell and T-cell development, have been implicated in gene arrangements involving TCR genes and in the SIL/SCL rearrangement, which involves two genes not normally rearranged. However, other mechanisms must clearly be active in generating these molecular abnormalities and perhaps they relate to the multistep maturation and differentiation processes and continuous cell turnover seen in hematopoietic cells. The difficulties in obtaining human solid tumor samples may make it more difficult to identify translocations involving TF genes in solid tumors. Recently, the cytogenetic analysis of solid tumors has improved and specific cytogenetic abnormalities have been associated with specific types of tumors. With advanced techniques, such as fluorescent in situ hybridization (a technique that does not depend on cell growth) and PCR, abnormalities involving TF genes will be discovered. Abnormalities of TF genes, other than translocations, have been seen in a broad variety of nonhematopoietic malignancies. The ***p53*** protein has been shown to bind DNA in a sequence-specific fashion and interact with a variety of DNA tumor virus oncoproteins. The broad range of cell

abnormalities will likely be implicated in many solid tumors. We have detailed several examples of how gene rearrangements that accompany chromosomal translocations in acute leukemia can alter the expression or activity of cellular TFs. Several translocations generate fusion RNA transcripts and fusion TF proteins with altered functional characteristics. Other translocations result in the expression of a gene not normally detectable in hematopoietic cells or alter the level of its expression, or affect the promoter usage or ***exon*** structure of the gene (Table 2). Studies are underway in many laboratories to characterize the biologic activity of these abnormal TFs and it remains to be proven that these molecular abnormalities are directly linked with leukemogenesis. The identification of abnormal fusion transcripts and proteins may allow specific therapies to be directed against "tumor-specific" DNA, mRNA, or protein targets. Therapeutic strategies based on ***antisense*** or ribozyme technology may be used to turn off expression of these genes and inhibit leukemia cell growth. Immunologic methods can also be used to direct therapy against the malignant cells.

L5 ANSWER 29 OF 32 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 94028468 MEDLINE
DOCUMENT NUMBER: 94028468 PubMed ID: 1340159
TITLE: Antisense RNA and p53 regulation in induced murine cell differentiation.
AUTHOR: Khochbin S; Brocard M P; Grunwald D; Lawrence J J
CORPORATE SOURCE: Laboratoire de Biologie Moleculaire du Cycle Cellulaire Unite INSERM 309, Centre d'Etudes Nucleaires de Grenoble, France.
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1992 Oct 28) 660 77-87.
Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199310
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19970203
Entered Medline: 19931022
AB ***p53*** expression is strongly modulated during the process of induced differentiation, at the same time as both cell cycle and genetic expression become modulated, giving rise to a commitment to terminal differentiation. We took advantage of two murine cell lines inducible for differentiation, an erythroleukemia and a melanoma cell line, to outline common features of the regulation of ***p53*** expression during the differentiation process. We found that ***p53*** mRNA decreased early after induced differentiation and that regulation was controlled at a posttranscriptional level. Our data showed that this regulation affects ***p53*** pre-mRNA maturation. Because, in both systems used, actinomycin D treatment abolished the inducer-mediated decrease of ***p53*** mRNA, we looked for induced RNAs potentially involved in this process. Using different parts of the ***p53*** gene and flanking regions as probes, we identified three RNA species whose expression is modulated during induced differentiation. A first species is made of high molecular weight RNAs that accumulate in the nuclear compartment and seem to represent ***antisense*** transcripts of the ***p53*** gene. A second species, 1.3-kb long, was found to accumulate in the nucleus of induced MEL cells and was homologous to a restricted part of the first ***intron*** of the ***p53*** gene due to the presence of a B1 repetitive element in an ***antisense*** orientation with respect to the ***p53*** pre-messenger RNA. Finally, a family of B2-containing RNAs was observed in both cytoplasmic and nuclear compartments. The variation in the amounts of sense and ***antisense*** RNAs, respectively, suggested an interesting speculative model for the maturation of B2-containing pre-messenger RNAs.

L5 ANSWER 30 OF 32 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1992:52757 CAPLUS
DOCUMENT NUMBER: 116:52757
TITLE: BstNI/NciI polymorphism of the human p53 gene (TP53)
AUTHOR(S): Chumakov, P. M.; Jenkins, J. R.
CORPORATE SOURCE: Engelhardt Inst. Mol. Biol., Moscow, 117984, USSR
SOURCE: Nucleic Acids Research (1991), 19(24), 6969
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English

p53 gene that can be revealed using restriction nuclease digestion of PCR-amplified DNA segment is reported. Left-hand (sense) oligonucleotide 5'-GTTGCCCAGGGTCCCCAGGCCTCTGATTCCTCACT-3' corresponded to the region of ***intron*** five 12 bp upstream of ***exon*** 6; Right-hand (***antisense***) oligonucleotide 5'-GGGAGGCCCTTAGCCTCGTAAGCTTCA-3' corresponded to the region of ***intron*** six 165 bp downstream of ***exon*** 6. The amplified fragment was purified by spermine pptn., digested with BstNI or NciI restriction nucleases and subjected to electrophoresis through 2% agarose. BstNI (CCAGG) and NciI (CCGGG) cleave different allele fragments of the ***p53*** gene: BstNI (K1): 206 + 130 bp; NciI (K2): 221 + 130 bp. Allele frequencies calcd. from 56 unrelated Caucasians were K1 = 0.31 and K2 = 0.69. The polymorphic A/G nucleotide responsible for BstNI/NciI restriction site polymorphism is localized in ***intron*** 6, 61 bp downstream of ***exon*** 6 of the ***p53*** gene (17p13). Mendelian inheritance was demonstrated in 2 three-generation families.

L5 ANSWER 31 OF 32 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1990:566707 CAPLUS
DOCUMENT NUMBER: 113:166707
TITLE: AccII polymorphism of the p53 gene
AUTHOR(S): De la Calle-Martin, Oscar; Fabregat, Virginia; Romero, Matilde; Soler, Jesus; Vives, Jordi; Yague, Jordi
CORPORATE SOURCE: Servei Immunol., Hosp. Clin., Barcelona, 08036, Spain
SOURCE: Nucleic Acids Research (1990), 18(16), 4963
CODEN: NARHAD; ISSN: 0305-1048
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A rapid and simple method is reported to analyze an AccII polymorphism within the human ***p53*** gene using the polymerase chain reaction. PCR Primers: The primer sequences corresponded to the 4th ***exon*** of the human ***p53*** gene: Sense oligo 5'-AATGGATGATTTGATGCTGTCCC-3' and ***Antisense*** oligo 5'-CGTGCAAGTCACAGACTTGGC-3'. An AccII (CGCG) digest of the amplified fragment identifies 2 alleles; A1 = 259 bp and A2 = 160 bp + 99 bp. Allele frequencies were calcd. from 90 unrelated Caucasians. A1 = 0.32 A2 = 0.68. The polymorphic AccII recognition site occurs within the 4th ***exon*** of the human ***p53*** locus (17q13).

L5 ANSWER 32 OF 32 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1990:71437 CAPLUS
DOCUMENT NUMBER: 112:71437
TITLE: An antisense RNA involved in p53 mRNA maturation in murine erythroleukemia cells induced to differentiate
AUTHOR(S): Khochbin, Saadi; Lawrence, Jean Jacques
CORPORATE SOURCE: Dep. Rech. Fondam., CEN-Grenoble, Fr.
SOURCE: EMBO Journal (1989), 8(13), 4107-14
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A post-transcriptional control of gene expression was found to be responsible for a down-regulation of p53 mRNA accompanying the induced differentiation of murine erythroleukemia (MEL) cells. Such a post-transcriptional control was governed by the induced synthesis of an RNA species (inRNA). In an attempt to find a potential candidate for such a function, the post-transcriptional regulation of p53 mRNA was localized in the nuclear compartment of the cells; then various fragments of the p53 gene were used as probes for induced RNA(s) susceptible to interacting with p53 pre-mRNA. This exptl. approach allowed for the identification of a nuclear RNA mol., .apprx.1.3 kb long, which was recognized specifically by a PstI-HindIII fragment located in the 5' part of the first intervening sequence of the p53 gene. This RNA accumulated when cells were treated by the inducer concomitantly with high mol. wt. p53 mRNA precursors. However this RNA was not a maturation product of p53 pre-mRNA as evidenced by its antisense orientation with respect to this RNA. Moreover it was markedly enriched in the poly(A)+ fraction. The complementary part of inRNA in the p53 gene has been sequenced over .apprx.1200 bp; no extensive homol. was found in gene data banks but 3 restricted areas of the sequence were found homologous to a limited no. of genes; they were themselves partially homologous to known B1 repetitive sequences. Possible implication of such a sequence in the regulation of p53 gene expression is discussed.

> kwic 2 4
WIC IS NOT A RECOGNIZED COMMAND
he previous command name entered was not recognized by the system.

"HELP COMMANDS" at an arrow prompt (=>).

=> d kwic 2 4

L5 ANSWER 2 OF 32 USPATFULL
DETD In the recombinant retroviral vectors, the orientation (a) vector showed higher levels of ***p53*** expression. It is contemplated that other retroviral promoters in the construct will suppress the .beta.-actin promoter, as described in other. . . all promoters are aligned in the same direction of transcription (Emerman & Temin, 1984). Another possible explanation is that the ***intron*** and its enhancer in the .beta.-actin promoter are spliced out of the retroviral message during the first round of retroviral. . . therefore this effect may have some degree of promoter specificity (Emerman & Temin, 1984; Gunning et al., 1987). If some ***antisense*** transcripts were produced in orientation (a), alternate transcripts should have been detected by Northern analysis. However, these transcripts were not detected. The effectiveness in expression of functional ***p53*** protein by the orientation (a) construct supports the absence of inhibition by ***antisense*** . The use of .beta.-actin promoter in orientation (b) with an LNL6 retrovirus yielded low rates of infectivity and low levels of gene expression (Owens & Boyd, 1991). Therefore, to maximize expression of ***p53*** , it may be advantageous to utilize different transcriptional orientations for the genes inserted in the retroviral vector.
DETD . . . the presence of 8 .mu.g/mL polybrene. This transinfection was repeated once daily for 3 days. To examine whether the transduced ***p53*** gene was expressed in these cells; the reverse transcription-PCR analysis used sense primers for .beta.-actin promoter sequences 5' to the promoter/ ***p53*** junctional sequences and an opposing ***p53*** cDNA ***antisense*** primer located within ***p53*** cDNA ***antisense*** primer located within ***p53*** ***exon*** 4. These primers are specific for the retrovirally transduced ***p53*** . PCR products were evaluated by Southern blot hybridization with a .sup.32p-labeled, nick-translated ***p53*** cDNA probe. A .beta.-actin/ ***p53*** segment was detected in H226Br cells transduced with wt- ***p53*** , whereas it was not present in parental and LNSX virus-infected cells. Western blot analysis demonstrated detectable levels of ***p53*** protein following LNp53B retroviral infection in ***p53*** -negative H358a cells.
DETD An ***antisense*** ***p53*** RNA probe was synthesized as above from a plasmid containing a ***p53*** CDNA template. Amplimers corresponding to ***exon*** 5 [5'-TACTCCCCTGCCCTCAACAAG-3' (SEQ ID NO:25)] and ***exon*** 8 [5'-CTTAGTGCTCCCTGGGGGCAG-3' (SEQ ID NO:26)] were used to amplify a 500-bp ***p53*** cDNA sequence by PCR from the complete ***p53*** CDNA. This sequence was subcloned into the pGEM-3zf(-) transcription vector (Promega Corp). The RNA probe was used in northern blot. . .
DETD . . . ID NO:27)] and another from the catalytic domain of the ribozyme sequence [5'-TCGTCCAAAAGGACTCATCAG-3' (SEQ ID NO:28)]. The level of endogenous ***p53*** expression was also assayed by RT-PCR using a primer corresponding to ***exon*** 1 [5'-GGGAGAAAACGTTAGGGTGTG-3' (SEQ ID NO:29)] and ***exon*** 4 [5'-TGCAAGTCACAGACTTGGCTG-3' (SEQ ID NO:9)] of ***p53*** . For northern blot analysis, the membrane was hybridized with an ***antisense*** ***p53*** RNA probe as described above. Hybridization and washing were performed according to the protocols supplied by Promega Corporation.

L5 ANSWER 4 OF 32 USPATFULL
DETD As demonstrated by the data shown herein and reported in Arora, 1998, the anti- ***p53*** ON suppress the expression of ***p53*** in post-hepatectomy rats. Morpholino and C-5-P ON's were found to be more effective than equivalent amounts of the unmodified phosphorothioate. . . liver. The morpholino oligomer is surprisingly effective in view of the fact that it is targeted at a coding region (***exon*** 10), well downstream of the AUG start region, the latter of which is the conventional target for RNAse-inactive ("steric blocker") ***antisense*** oligonucleotides.

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